The
Southern Surgeon

Subscription in the United States, $8.00

Vol. XVI, No. 7 Copyright 1950 by
The Southern Surgeon Publishing Co. July, 1950

PROLAPSED GASTRIC MUCOSA

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PROLAPSE of the gastric mucosa is generally due to the presence of a stalky tumor. In rare instances, it may result entirely from an abnormal redundance and relaxation of the mucous membrane in the pyloric region of the stomach. This redundant mucosa, as in our case, may enter the first part of the duodenum and completely though intermittently occlude the pyloric lumen.

This condition seems to be a rare one to judge by the number of cases in the literature. In a review of the literature, Harris and Byrne[1] found 27 cases and reported 9 more. Redundant gastric mucosa has been reported to occur in from 0.23 per cent to 3.38 per cent of all patients with gastro-intestinal tract disease,[2 3] although all cases are not of clinical significance. The precise etiology is unknown. Eliason and Wright[4] thought a local hypertrophy of the gastric mucosa followed a low grade inflammation, and that these hypertrophied folds of gastric mucosa were swept through the pylorus by peristalsis. Rees[2] felt that a narrowing of the pyloric ring preceded the hypertrophy of the gastric mucosa. In the effort to force the gastric contents through this abnormally nar-

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Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are a result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

rowed opening, the mucosa is loosened, mobilized and gradually drawn through the pyloric ring, according to this author. Since Harris and Byrne[1] noted an hypertrophy of the pylorus at operation, they felt that spasticity or hypertrophy of the pyloric musculature might well be the basic factor in the causation of this entity. We feel that this condition is not as unusual as it is presumed to be, and that the diagnosis will be made more frequently as it becomes better known, though to our knowledge this is the 37th case reported where the diagnosis has been substantiated by surgery.

Symptoms are characteristically intermittent, non-specific and of long duration. The patient may offer the following complaints:

1. vague epigastric discomfort.
2. severe episodic pain between meals which is not relieved by food, but indeed may be commonly aggravated by eating, or aggravated or brought on by bodily exertion.
3. vomiting.
4. hematemesis and/or melena.
5. weight loss as a logical consequence of these symptoms.

Cove and Curphey[8] have reported 2 cases where a tumor mass in the region of the pylorus, soft and mobile, could be palpated. This finding was not present in the case reported here. The only other physical finding of any consequence is tenderness in the pyloric region which may be associated with numerous lesions in this area.

As a general rule, examination of the blood and urine reveals no abnormality. Due to prolonged intermittent bleeding, secondary anemia may be found in some patients. Scott[6] reports that in over two-thirds of the patients gastric analyses show acid values within normal limits. Occult blood may be found in the stools of many of these patients.

Little is known of the value of gastroscopy in these cases, but it may be of value in eliminating other gastric lesions.

The fluoroscopic appearance and the findings on the x-ray films are quite characteristic. Kirklin[7] has pointed out the mushrooming or cauliflower effect of the gastric mucosa in the first portion of the duodenum which can be demonstrated by barium studies. This defect is more readily shown in the horizontal than in the erect position. This filling defect in the base of the bulb is often lobulated, and the continuity of these markings with the gastric rugae can frequently be seen. Evidence of gastric rugae in the first portion of the duodenum must be present to satisfy fully our diagnostic criteria for true prolapse. The prolapse can be reduced under fluoroscopy in some instances by pressure over the bulb. In uncom-

plicated cases, the duodenal bulb appears normal except for the filling defect at its base, and spasm or irritability are usually not present. Despite these individualistic features, the diagnosis may be easily missed by the roentgenologist if he does not keep this entity constantly in mind.

Case Report

This 56 year old white male entered the hospital on Oct. 19, 1949, with a chief complaint of intermittent epigastric pain radiating through to the back of two months' duration. When this pain became intense, it was accompanied by nausea and vomiting. His present exacerbation began with a burning, crampy, epigastric pain, constantly present but varying in intensity. This pain was relieved momentarily by milk and antacids, but soon became aggravated by these substances or by any foods. There was a ten pound weight loss during this two month period.

Since 1920, this man had had intermittent bouts of pain, followed by nausea and vomiting. In the early years of his illness he frequently vomited small amounts of blood and mucus, and noticed tarry stools for months at a time.

In 1935, he was hospitalized, had a gastro-intestinal series within normal limits, but was told he had a duodenal ulcer. In 1947, he was again hospitalized with complaints of mid-epigastric pain, nausea and vomiting. A gastro-intestinal series at that time showed "a small, spastic, deformed duodenal cap" but no ulcer niche or crater was demonstrated. Nevertheless, a diagnosis of chronic duodenal ulcer was made and the patient discharged with a complete ulcer regime.

Physical examination at the present admission showed a thin, elderly man, appearing older than his chronological age and showing evidences of recent weight loss. Blood pressure was 135/75, pulse 80 per minute and respirations 14 per minute. There was extreme tenderness in the mid-epigastrium and moderate voluntary rigidity. There was a left indirect inguinal hernia, which was easily reducible, and a smooth, diffusely enlarged prostate gland. The examination was otherwise within normal limits.

Laboratory examinations were negative.

A gastro-intestinal series was performed on October 26 and repeated on November 22. These examinations revealed essentially the same findings. The esophagus was normal to fluoroscopy. There was considerable spasm and irritability in the antral region of the stomach producing some inconstant deformity of its distal one-third. The prepyloric rugae did not appear thickened.

There was a smooth filling defect at the base of the duodenal bulb producing a mushroom type of deformity. The markings at the base were continuous with the gastric rugae. These findings were considered due to herniated gastric mucosa. Pressure over the bulb reduced this herniation. At the next peristaltic wave, however, this hernia recurred. In the immediate post-bulbar portion of the duodenum, there was a spastic narrowing of the lumen. There was no ulcer crater in the bulb or post-bulbar region. Figure 1 shows the typical x-ray features.

X-rays of the chest were within normal limits.

Fig. 1. Note the large filling defect in the base of the duodenal bulb and the gastric rugae extending into this defect. The narrowed, spastic postbulbar segment is well shown.

Gastroscopy was not performed because of an old fracture of a cervical vertebra.

The patient was placed on a strict medical regime including bed rest, phenobarbital, atropine, and small hourly feedings, with antacids on the half hour. Despite this rigid program his pain failed to subside.

He was seen by one of us (W. J. C.) in consultation, and surgery was advised. Accordingly on Dec. 9, 1949, under continuous spinal anesthesia, a left rectus muscle-splitting incision was used to open the abdomen. No evidence of ulcer, residual scars or adhesions were found. However, the pylorus appeared considerably thicker than normal, and on palpation, the first portion of the duodenum seemed to contain a definite but poorly outlined mass, not at all like a pedunculated tumor but giving a doughy feel, a sensation of being unable to compress the duodenum completely. Therefore, the first portion of the duodenum was incised in the direction of its long axis. There was an immediate outpouching of redundant gastric mucosa. The operator then introduced a finger into the duodenum through this opening. There was an abundance of mucous membrane proximally and distally. No evidence of ulcer could be made out either by this palpation or by inspection.

The duodenum was then closed and a routine resection of the distal two-thirds of the stomach performed. A Polya technic with an anterior gastrojejunal anastamosis in two layers was performed. The abdomen was closed in layers in the usual fashion, and three retention sutures of number thirty-two stainless steel wire were passed through all layers anterior to the posterior rectus fascia and tied with buttons.

The patient stood the procedure well and had an uneventful postoperative course.

The specimen was examined grossly before fixation. The most striking finding was the redundancy of the mucosa which was thrown into great transverse folds in the pyloric region of the stomach, some of these folds measuring 8 cm. in height. With the stomach opened, and looking down through the intact pylorus (fig. 2), the pylorus was seen to be completely obstructed by

Fig. 2. Note the circular folds of redundant gastric mucosa shown in the upper part of the photograph which completely close the pyloric exit of the stomach.

folds of gastric mucosa which extended through the ring into the duodenum, but one could put a finger through the pylorus into the duodenum by pushing aside the great folds of mucosa. The mucous coat appeared to be loosely attached and extremely mobile. Figure 3 shows the abnormal mobility of the mucous coat.

Histologic examination of sections taken from the pylorus and fundus revealed a similar picture. The normal mucosal pattern was maintained. There was slight hyperplasia of the mucus-secreting surface glands and normal appearing gastric glands. The usual lymphocytic infiltration in the lamina propria was not increased. The muscularis mucosae of the portions of redundant mucosa was conspicuously prominent. The submucosa was edematous and loose connective tissue widely separated mucosa and muscularis. The blood vessels in the sub-mucosa were prominent and distended by erythrocytes, and the perivascular connective tissue was intensely hyperemic.

The muscular wall was composed of three distinct layers of hypertrophied muscle. There was edema and separation of the individual muscle bundles and a mild lymphocytic infiltration. The sub-serosal lymphatic channels were

Fig. 3. The abnormal mobility of the prepyloric gastric mucosa is demonstrated.

Fig. 4. Fundus. The submucosa is loose and edematous with prominent blood vessels. The muscularis is hypertrophied.

Fig. 5. Pylorus. Note the redundant mucosa, loose submucosa and hypertrophied muscularis.

ectatic and incidental polymorphonuclear leukocytes were dispersed here and there. Figures 4 and 5 show these features well.

Comment

The abnormal relaxation and redundance of the gastric mucosa must be considered the pathologico-anatomic basis for this disease and therefore the term "relaxed gastric mucosa" may be preferred to "prolapse of the gastric mucosa." The symptoms in the case described were the result of a true prolapse through the pylorus into the duodenum. But in other instances, as in the first recorded case,[8] the complaints may be the result of a valve-like action of a transverse fold of mucous membrane intermittently closing off the pyloric exit of the stomach. It would seem that "relaxed gastric mucosa" is a preferable general term, whereas "prolapsed gastric mucosa" should be restricted to cases such as the one presented here where an actual extrusion of mucosa into the duodenum is demonstrated.

The accompanying pyloric hypertrophy is probably a result of the increased effort of drawing the gastric mucosa into the duodenum. This continuous traction tends to stretch and loosen the mucosa still more.

Treatment

Treatment of the vast majority of these cases may be confined to medical measures. We feel that all symptomatic patients in whom the diagnosis is made by history and by x-ray examination should have an adequate test of medical therapy. If such a regime does not succeed in returning the patient to his former active life without rigid adherence to a limited diet and antispasmodics, we feel surgery should be advised. Patients who have persistent pain, nausea and vomiting, or hemorrhage after an adequate trial of medical treatment we believe should have surgery. Although the etiology of this lesion is not clear, the pathology is on a purely mechanical basis. Once this entity is well established, the patient will in all probability have no permanent relief short of mechanical correction. In the past, a variety of procedures have been used in the surgical treatment of this condition. They have ranged from gastrotomy, excision of the mucosa with suture of the remaining mucosa to the underlying muscular layer, through pyloroplasty and gastro-enterostomy to gastric resection. In planning the surgical approach to such a case, one must concede that this condition per se is a progressive one, and that the surgeon must treat not only the existing pathology, but also make an effort to prevent a recurrence. We feel that gastric resection best accomplishes these ends. We feel too that a Polya type of resection, because of its wide stoma, is perhaps the best type of resection.

The doughy sensation on palpating the pylorus and duodenum and the accompanying hypertrophy of the pylorus are significant

findings at operation. The demonstration of gastric mucosa in the duodenum by opening this structure is diagnostic. We feel that before the resection is started, in each case where the diagnosis is suspected, the duodenum should be opened first to confirm the diagnosis in the manner described.

Unless acid values are high, we consider it unnecessary to remove the usual four-fifths of the stomach, as one would do routinely in a gastrectomy performed for duodenal ulcer.

SUMMARY

1. A case of symptomatic prolapse of the gastric mucosa through the pylorus, confirmed by surgery and pathological examination of the specimen, is reported.

2. The incidence, etiology, clinical picture, diagnosis and treatment of this condition are briefly reviewed.

3. A sub-total gastrectomy, with a preliminary duodenotomy to confirm the diagnosis, seems to be the treatment of choice.

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POLYPOID LESIONS OF THE COLON

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POLYPOID lesions of the colon may be single or multiple; and the proper treatment depends upon the type, location, size, mobility, consistency and biopsy findings.

Any study of this condition should take into consideration a workable classification of colonic polyps, and three such may be of value: First, that suggested by Bockus of—

I. Ademona of colon
  A. Single (adult [acquired] type)
  B. Multiple
    1. Adult (acquired) type
    2. Adolescent (congenital disseminated type), multiple polyposis, heredofamilial polyposis, multiple adenomatosis, disseminated polyposis, etc.
      a) Segmental
      b) Disseminated
        1) Without diffuse hyperplasia
        2) With diffuse hyperplasia
  C. Papilloma (papillary adenoma, villous polyps, etc.)

or—

II. that of Erdman and Morris—
  A. Adult (acquired) type
  B. Adolescent (congenital) disseminated type

or—

III. that of Wesson and Bargen—
  A. Post-inflammatory (pseudopolyps)
  B. True polyps.

Of these various classifications the first is the most valuable for all purposes.

The incidence of the various forms of colonic polyps is rather common and one should always be alert as to the possibility of their existence. As a consequence of this alertness one should never consider any examination of a patient complete until digital rectal and proctoscopic examinations have been made. Bargen has estimated

Read before The Southwestern Surgical Congress, Houston, Texas, Sept. 27, 1949.

that some form of polypoid hyperplasia in the colon is present in 50 per cent of all people over 30 years of age. Necropsy studies, however, reveal an incidence range varying from 2 to 21 per cent. Buie and Martin in sigmoidoscopic examinations of a large group of patients with symptoms of colonic dysfunction, found 4 per cent to have polyps. C. H. Mayo found 1 of every 55 patients, where x-rays of the colon were taken, to have demonstrable polyps. In chronic ulcerative colitis Bargen reported that 16.1 per cent of 871 cases revealed the presence of polypoid disease. Various studies of carcinoma of the colon have revealed associated polypoid disease in 17 to 76 per cent of the patients. The incidence of multiple polypoid disease of the colon (congenital) is rather rare and is reported variously as by Cattell as constituting 0.5 per cent of patients with large bowel lesions coming to operation at the Lahey Clinic down to 0.04 per cent reported by Mayo and Wakefield as determined in a group where one or more polyps could be visualized by sigmoidoscopy.

Etiology

The etiology of polypoid lesions of the colon is often obscure or undeterminable. However, a number of factors apparently play an important role in the production of the disease in certain instances. These factors include: 1) Inflammation, as in chronic ulcerative colitis where a high incidence of polypoid disease subsequently develops or perhaps in the inflammation produced by the ova of certain parasites, as oxyuris vermicularis. 2) Chronic irritation as produced by constant minute traumas on a receptive field. 3) Primary epithelial hyperplasia. Saint and Dukes hold that the basic change consists of independent exorbitant hyperplasia in one or more areas of mucosal epithelium. 4) Primary subepithelial changes. Fowler and Bargen in 1940 claimed that the primary changes occur in the subepithelial structures, especially in the lymphoid tissues. 5) Hereditary predisposition. Cripps first described the familial features of the multiple type—the condition occurs as a dominant mendelian characteristic; and 6) embryonal defects as manifested by misplaced embryonal rests are occasionally important etiologically.

Pathology

Pathologically polyps show a distinct tendency to increase progressively in frequency in a caudal direction through the intestinal tract. Rankin and Grimes estimate the frequency to be eight times more common in the rectosigmoid and rectum than in any other section of the colon. In ulcerative colitis associated polyps occur in the rectum in over 50 per cent of cases where they develop. Of

solitary polyps, 65 to 75 per cent occur in the rectum or rectosigmoid and can be seen with a scope.

In gross appearance 35 to 45 per cent will be multiple whereas the others occur singly. The types will be pedunculated, sessile or villous; and the polyps may be ulcerated, inflamed, fixed or free of all these characteristics. The color varies from pale pink to deep red.

Microscopically the typical polyp has 1) a stalk derived from and continuous with the submucous fibrous tissue and 2) an epithelial covering continuous over the pedicle and with the epithelium of the adjacent bowel. Malignant change with or without invasion of the base may be present. The surface of a polyp may appear malignant and the base be non-malignant.

Clinical Features

A polypoid lesion may be present in the colon for years without symptoms. Proctologists in reporting large series find 50 to 75 per cent of those encountered on routine examinations to be asymptomatic.

The symptoms which may occur are dependent on, 1) the size of the lesions, 2) the situation of the lesion, 3) the number of polyps present, 4) the amount of the colon involved, and 5) the presence or absence of complications.

The condition is predominantly a disease of middle age, or older, except in the heredofamilial type where the average age is 20 years. The majority of the patients are 40 or over, although such an entity may occur in infancy.

The sex incidence is approximately three males to two females.

The disease is said to be more common in the white race than in the colored races.

The duration of illness before discovery—other than in those patients where discovery is an incidental finding during examination—varies widely, but probably at least 50 per cent of those with symptoms will have had such symptoms for a period of 3 to 10 years or more.

The heredofamilial trait is definite in the multiple polypoid type of disease (other than those secondary to ulcerative colitis). McKenney made an interesting study of three such family groups involving 52 persons, of which 40 per cent had this type of disturbance.

The symptoms most commonly encountered are: 1) Blood in the stools occurs in about two thirds of the patients. This is usually

intermittent and small in amount—and may be most often noted immediately after a bowel movement. 2) Diarrhea is reported in one out of six patients. At times acute exacerbations of this diarrhea may occur. Excessive mucous with the stools may be present in some individuals. 3) Abdominal pain—characterized most commonly by a vague general distress—occurs in about 25 per cent; this may be cramplike at times but if so is seldom severe. This pain is usually increased during and immediately after a bowel movement. 4) Painful or difficult defecation characterized by tenesmus, urgency or sense of incomplete evacuation occurs occasionally.)5 A protruding mass through the anal opening is noted rarely. 6) The spontaneous expulsion of a tumor mass is a rare occurrence, as a pedunculated polyp does not often pull off of its stalk.

The clinical course of the disease may vary greatly. The condition may persist for years without symptomatology and then change rather suddenly due to the development of complications.

Examination of these patients should be complete and must include: 1) Physical examination may reveal no significant findings. Digital examination of the rectum, however, will demonstrate a palpable polyp or multiple polyps in about 50 per cent of such patients. 2) Laboratory examination often contributes little helpful information. However, such an examination is always necessary. A mild anemia may be encountered. Occult blood will probably be found in two out of three such patients if all stools are checked. Any change in the sedimentation rate is dependent on the presence of infection or ulceration and this is true also for the presence of leukocytosis. 3) Proctosigmoidoscopic examination to 25 cm. will demonstrate the presence of one or more polyps in two thirds to three fourths of all patients with this disease. One cannot urge too strongly the inclusion of a proctoscopic examination in all routine examinations as so many of these silent polyps will be demonstrated by this examination alone. 4) X-ray examination complements proctosigmoidoscopy as lesions of 1 cm. or less in diameter will usually be obscured by the barium enema and as a result many lesions missed by the x-ray will be visible with the use of the scope. One must remember, however, that all such lesions beyond the reach of the sigmoidoscope can be recognized only by means of the x-ray. This examination should consist of a routine barium enema first, followed by a barium-air double contrast enema. By means of this method polypoid tumors will be visualized as projections into the intestinal lumen which stand out in sharp contrast to the air-distended bowel. It may be necessary to repeat the x-ray examinations several times if bleeding persists and nothing has been found on the previous examinations on which to make a diagnosis.

The complications most often encountered in this disease are: 1) Inflammation or ulceration characterized by more marked diarrhea, blood, pus, pain, anorexia and weight loss. 2) Obstruction of the bowel may bevelop. This may be transient (intermittent) at times. When this occurs the usual symptoms of intestinal obstruction are present: i.e. pain, obstipation, distention, nausea, vomiting, etc. 3) Malignant change is not unusual and is one of the most serious complications which must ever be kept in mind. Vernon David has stated that he believes that wherever such a change is present the condition was malignant from the beginning. In large groups of statistics the incidence of malignant change varies from 5 to 85 per cent of all such patients, and this wide variation is due no doubt to a varied interpretation of the pathologic findings. As indications of malignant change, the presence of the following gross characteristics must be taken into consideration: general gross appearance, ulceration, nodular irregularity, fixation and induration. Microscopically the entire lesion must be examined, especially to determine if there has been any invasion of the base in those instances where the adenomatous portion appears to show early malignant changes. One must remember that polyposis has a close relationship to carcinoma of the colon—also that polyposis occurring with chronic ulcerative colitis often shows a malignant change. In heredofamilial polyposis sooner or later nearly every individual with the disease develops malignancy of the colon. Mayo and Wakefield estimate that one third of this latter group have carcinoma of the colon at the time the primary diagnosis is made.

Diagnosis

The diagnosis is thus found to be dependent primarily on the digital, proctosigmoidoscopic and x-ray examinations. One must always determine whether the lesions are single or multiple. Biopsy examination is advisable, but one must remember that one can't determine benignancy from a single specimen. Whenever polyps are found one should always look for carcinoma, diverticulitis and ulcerative colitis. In the heredofamilial type one should always advise that the other members of the patient's family should be checked.

Differential diagnosis must rule out carcinoma, idiopathic ulcerative colitis, amebic and bacillary dysentery, diverticulitis and anorectal disease. Do not forget that any or all of the above conditions may coexist with polypoid lesions of the colon.

Treatment

In the treatment of polypoid lesions of the colon the following

basic principles must be kept in mind always: 1) The proneness of a given tumor to become malignant cannot be predicted with certainty. The time required for such a change is unknown. One should look on every lesion as potentially malignant and eradicate it promptly on discovery. 2) The malignant potentiality is no less even if there are no symptoms; hence, destroy the lesion. 3) The removal of one or more lesions gives no assurance that others will not appear. As long as tumor-bearing tissue is present new lesions may develop. As a result these patients must be reexamined regularly for long periods of time.

Medical management does not cure the condition. The use of low residue diet and other therapeutic measures is of little value.

X-ray therapy has been utilized by McKinney, Van Zant and others. These investigators have reported some beneficial results but cure by such means is uncertain and extremely doubtful.

Surgical management of polypoid lesions of the colon offers the best outlook to these patients. The type of treatment varies with the location and type of disease present. The following procedures are the most useful:

1) Fulguration is applicable to lesions accessible to the sigmoidoscope. Pedunculated polyps and sessile polyps on the posterior surface below the peritoneal reflection are most suitable for this form of therapy. The two most dangerous complications are perforation (most apt to occur when lesion is on the anterior surface above the peritoneal fold) and hemorrhage (apt to occur when the slough develops) which is rare. If the lesions are low these patients remain ambulatory during treatment. Large lesions may require one or more fulgurations. The site of the fulguration should be at the junction of the body and pedicle—but if this is impossible initially, destroy the body first. It is wise to believe that more tissue is destroyed than appears to be the case. Repeated observation is essential till slough has occurred and healing advanced.

2) Ligation and excision may be done when there is a well marked stalk in the case of an isolated polyp. Sometimes this is combined with fulguration of the base. The electric snare is employed by some operators.

3) Transcolonic excision is the procedure of choice where there is one or more indiscrete polyps with stalks high in the colon.

4) Segmental resection is advisable in instances of sessile polyps which cannot be fulgurated or where the base of a polyp has become fixed to the bowel.

5) Ileostomy was formerly used by many who thought that re-

gression would occur in disseminated polyposis. Its use in polypoid disease is now usually confined to that of a preliminary step in extensive colonic resection in polyposis secondary to chronic ulcerative colitis or heredofamilial polyposis.

6) Ileostomy with total colectomy is valuable in generalized polypoid disease of the colon. Cattell often prefers this form of surgical management.

7) Ileosigmoidostomy (or ileoproctostomy) with subtotal colectomy is the form of surgical therapy preferred by many. In carrying out this procedure one must first destroy all the polyps in the sigmoid and rectum with fulguration, later doing an ileosigmoidostomy and subsequently a subtotal colectomy. These last two steps are done separately or at one stage depending on individual circumstances.

Prophylactic treatment consists of always being on the lookout for the presence of polyps and adequately treating those found.

Prognosis

The prognosis is dependent on the location and type of lesion present. Mayo and Smith report two deaths in 62 cases where transcolonic resections were done. Rankin and Grimes report a 15 per cent mortality in colectomy in stages. Hullsiek found 35.1 per cent mortality in 51 cases of multiple polypoid disease. All such patients must be reexamined at regular intervals for at least five years and all new polypoid formations found must be destroyed.

The following illustrative cases are presented as examples of some of the various types of polyps and the associated therapy:

Case 1. Mrs. L. P. (a registered nurse), age 25 years. Three weeks prior to delivery of first pregnancy her obstetrician discerned a large pedunculated polyp on the anterior wall of the rectum about 4 cm. inside the anal opening. No symptoms referable to same. On Jan. 1, 1949, at the completion of delivery, the polyp was ligated and excised. Pathologic report was adenocarcinoma, grade I (adenomatous papillary polyp) without invasion of the base. Subsequent barium and proctoscopic examinations were otherwise negative. On January 20 under general anesthesia the site of the attachment of the pedicle was fulgurated. She has been examined every three months since and on Sept. 19, 1949, was still free of any evidence of further disease.

Case 2. Mrs. H. B., aged 60, ordained minister. General examination on Oct. 27, 1948, revealed a large sessile polyp about 6 cm. in diameter 7 cm. above anal opening. Only complaint was nervousness and occasional mild cramping in left abdomen. Admitted to hospital on November 10 and on the 20th combined abdominoperineal resection of rectosigmoid done. Pathologic report was adenocarcinoma (polypoid), grade I, of rectum. No metastases. Last seen on Sept. 1, 1949, at which time there was no evidence of recurrence.

CASE 3. Mr. L. L. H., aged 41, banker. First seen on Feb. 24, 1949, for a general check-up with complaint of hemorrhoids for many years with occasional bleeding. Examination revealed both internal and external hemorrhoids. X-ray studies reported "a polyp in the descending colon and another in the region of the splenic flexure." Admitted to hospital on March 10, and on March 15 a transcolonic excision of the two polyps was done. The pathologic report was benign adenoma (papillary) of the colon. Good postoperative course. Last seen on April 21, 1949, at which time he was doing nicely.

CASE 4. Mr. L. A. W., aged 65, grain elevator manager. First seen on April 7, 1948, with complaint of blood in stools. X-ray revealed two polyps in the descending colon on long stalks. Diverticuli were also present in the left colon. Entered hospital on May 5 and on the 10th a transcolonic excision of the two polyps was done. Pathologic report was adenocarcinoma, grade I, without invasion of the base. Postoperatively he had a rather stormy convalescence with multiple infarcts but finally left hospital on July 28. When last seen on Aug. 18, 1949, there was no evidence of recurrence and no further bleeding.

CASE 5. Mr. N. A. S., aged 56 years, rancher. In December, 1948, admitted with complete pyloric obstruction due to old ulcer. Also had marked adrenal insufficiency of long standing (Addison's disease). For about two months he had had some cramping in lower left abdomen and bleeding at intervals from the rectum. X-ray revealed complete pyloric obstruction and a polyp in the sigmoid. On December 27 a partial gastric resection (posterior Polya) was done. On April 25, 1949, he was operated on again after readmission because of further rectal bleeding and partial upper abdominal obstruction. Lysis of adhesions in left upper quadrant was done and a transcolonic excision of the sigmoid polyp was done (pathologically benign). Subsequently on June 18 the patient was again operated on for upper abdominal obstruction, at which time the previous anastomosis between stump of stomach and jejunum was broken down. More tissue was resected and a Roux Y anastomosis done. On Aug. 3, 1949, the patient was last seen and he was in good condition.

CASE 6. Mr. W. C., 21 year old veteran, was first seen in March, 1944, with ulcerative colitis which had been present for one year. Medical treatment was without benefit. On April 19, 1944, an ileostomy was done. Moderate improvement after this with weight gain of 20 pounds. Seen periodically after this. Readmitted to hospital on July 5, 1948, for reevaluation, at which time x-ray and proctoscopic examinations revealed marked chronic ulcerative colitis. A definite neuropsychiatric disturbance was also present. He was transferred to the surgical service on August 13 and on September 13 a total colectomy by the method described by Ravitch, in which the anal sphincter is preserved and the ileostomy brought out through this opening, was done. (The pathologic report was multiple polyposis and chronic ulcerative colitis.) Postoperatively he had a satisfactory course, except that there was some irritation about the anal opening. In three months' time he was passing semi-solid fecal material, and had partial control of these movements. There was considerable granulation in tract and it was necessary to dilate tract from time to time. On Jan. 20, 1949, he was doing well except that his mental attitude was still unimproved. He was last seen in May, 1949, when he was still mentally unstable although he had some improvement in the sphincter control.

CASE 7. Mr. T. R. S., aged 23, a veteran student, was admitted to the hospital on Dec. 26, 1946 with complaint of lower abdominal cramps, with

passage of blood and clots and two or three bowel movements daily. This had occurred intermittently for two years. His mother had died of carcinoma of the rectum in her early thirties and an aunt had died at 36 years of carcinoma of the bowel. Physical, proctoscopic and x-ray examinations revealed the presence of multiple polyposis. During the next ten weeks repeated fulgurations of the polyps in the rectosigmoid were done. On March 18, 1947, laparotomy with side-to-side ileoproctostomy was done. On April 10 a subtotal colectomy was carried out. (The pathologic report was congenital multiple polyposis, benign.) Postoperatively he has been proctoscoped every three months and fulgurations of any recurrent sessile polyps in the rectum have been done. He was last seen in July, 1949, at which time he was doing well with two or three soft stools daily.

CASE 8. Mr. A. M., a 34 year old colored veteran, was admitted to hospital on June 9, 1948, because of severe bleeding from rectum. Family history indefinite. History of recurrent diarrhea for four years. He was dehydrated and acutely ill, had a moderate anemia and Endamoeba histolytica were found in the stools. Proctoscopic and x-ray studies revealed multiple polyposis (extensive) of entire colon and biopsy of several of the polyps revealed carcinomatous changes. He was treated with carbasone and emetine and surgery was advised. On Sept. 8, 1948, a combined abdominoperineal resection of sigmoid and rectum was carried out and on September 27 the remainder of the colon was removed and an ileostomy done. Postoperatively he developed a venous thrombosis and the common iliac veins were ligated. The pathologic report was multiple polyposis with numerous areas of carcinomatous change, especially in the rectosigmoid and hepatic flexure regions. When heard from in May, 1949, no recurrence was evident.

CONCLUSIONS

1. A brief résumé of the various polypoid lesions of the colon has been presented, together with eight illustrative case histories.

2. A routine examination should include digital rectal, proctosigmoidoscopic, and x-ray studies.

3. The location, type and multiplicity of the lesions are important in treatment and prognosis.

4. A given tumor's proneness to become malignant cannot be predicted with certainty. Hence look on every lesion as potentially malignant and eradicate it promptly on discovery.

5. Surgical treatment is essential and repeated reexaminations at regular intervals for years is necessary. Destroy all new lesions that appear.

The prognosis in multiple heredofamilial polyposis is discouraging.

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SOME RESULTS OF ARTERIAL SPASM*

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THE purpose of this paper is to emphasize that continued or recurrent arteriospasm may result in pathologic changes in the arteries of the involved extremity. Often the original injury or lesion may be underestimated, and, unless the physician is aware of the ill effects of arterial spasm, proper treatment may be too long delayed. A year ago, Pomerantz[1] presented a full classification of vascular diseases and reviewed many of the experimental and clinical articles dealing with vascular spasm.

Leriche[2] defined spasm as "vasoconstriction exceeding the limits of normal, and giving rise to a pathological condition." He enumerated the following causes of vasospasm:

1. Cold.
2. Abnormal rubbing of an artery against some rigid object.
3. Distention of an artery by an embolus.
4. Thrombosis of an artery or a vein.
5. "All forms of excitation which may affect nerves of the cerebro-spinal system, especially after these have been wounded."

The last cause would include all forms of trauma and possibly emotional causes as well.

Vasospasm should be suspected in an extremity when some of the following signs and symptoms are present in varying degrees:

1. Cold sensitivity.
2. Coolness.
3. Cyanosis or color changes.
4. Hyperhidrosis.
5. Diminished or absent pulsations.

Some examples of different causes of arteriospasm are presented.

CASE 1. Anna W., white female, unmarried, aged 19, noted onset of pain and tingling in fingers and toes approximately one year before admission. This started with exposure to cold, and she had episodes of pain with pallor, cyanosis, and, later, rubor of both fingers and toes. Close questioning failed to reveal any emotional disturbance. In September, 1948, she had visited in Michigan. Following exposure to cold one evening, she developed small ulcers on the tips of her right first three fingers. Pain became more severe and attacks more frequent, so that during the past winter she had spent most of the day huddled near a fire. Menstrual periods were normal and she never had used tobacco.

*Read before the seventeenth annual Postgraduate Surgical Assembly of The Southeastern Surgical Congress, Biloxi, Miss., May 24, 1949.

Examination revealed a well developed girl of 19 who gave the impression of being quite stable emotionally. All peripheral pulses were weaker than normal (B. P. 132/74). There were recently healed lesions of several finger tips on the right hand, and definite sclerodermatous changes in both upper extremities. When placed in a room with temperature of 75 degrees F., her fingers and toes became cyanotic, with more pallor than redness. When her hands were placed in cold water there was marked blanching, especially of previously involved fingers.

There was marked flushing and a very significant rise in skin temperatures following the intramuscular injection of 75 mg. of Priscol (Ciba).

Operation was delayed for a month, and, during that time, she received an oral sympatholytic and adrenolytic compound being investigated clinically for Parke, Davis & Company. This agent has been designated as SY-28. This afforded her some relief from pain in her fingers and prevented the triphasic color changes on exposure to moderate cold.

In February, 1949, bilateral dorsal sympathectomy, as advocated by Smithwick, was done, with good immediate results. There was rapid disappearance of the scarred areas over the finger tips, and the ferric chloride-tannic acid sweating test described by Silverman and Powell[6] showed absence of sweating after exposure to heat lamps.

Comment

Lewis[3] found increased intimal thickening in advanced cases of Raynaud's disease, but, in comparable cases, Allen[4] could find no significant organic changes from arteriographic studies. Intimal thickening is found in varying degrees in all persons over fifty. In younger individuals with Raynaud's disease the intimal changes are probably the result of vasospasm.

Hypersensitivity to cold may not cause the typical picture of Raynaud's disease, but may be disabling, and, when long continued, can result in arterial disease. Two patients have been seen recently who were unable to work because of two or more of the signs of marked vasospasm. During the last war the effects of cold and moisture on the lower extremities of soldiers were widely observed, and sympathectomy proved of value in late sequelae characterized by cold, clammy, hyperhidrotic feet, whose pulsations were often weak or absent.[5] Only the most severe cases of hypersensitivity to cold require sympathectomy. Kleinsasser[14] found that sensitivity to even moderate cold was the predominant feature in a group of patients with vasospasm severe enough to require sympathectomy.

Case 2. Mrs. Elsie B., white, aged 55 (Courtesy of Dr. Barney Brooks), was first seen in the Medical Out-Patient Department of Vanderbilt University Hospital in January, 1937, complaining of pains in her shoulders, arms and knees. Roentgenograms showed hypertrophic arthritis of the cervical spine and a cervical rib on the right side. Brachial blood pressure and radial pulses were normal and equal.

The patient was next seen 7 years later, in August, 1944. For the preceding six months she had had increasing numbness, pallor and coolness of the right ring and little fingers. This was first noted while driving a car, and was aggravated by elevation of the arm. The numbness and tingling were relieved by exercise. One month before admission she scratched the tip of the right index finger and a "blood blister" developed, which failed to heal. Her symptoms were not influenced by cold.

Examination revealed blood pressure absent on the right, 140/80 on the left. There was slight prominence of the right supraclavicular fossa, with the right anterior scalenus muscle being more prominent than the one on the left. There was a strong pulsation of the right subclavian artery at this site, though the brachial and radial pulses were not felt. The pulsation stopped abruptly where the artery dipped under the clavicle. There was dry gangrene of the tip of the right index finger. There was no atrophy or anesthesia of the hand. Moszkowicz's test on the right showed a blush which returned slowly at an almost equal rate to all points beyond the level of the tourniquet. This suggested a definite diminution in the size of the lumen of the primary artery. Immersion of the hand in cold water produced no significant changes.

Operation was performed on Sept. 6, 1944.

Operative Note

"Previous to operation patient presented a very remarkable picture, almost characteristic of Raynaud's disease. X-ray showed presence of cervical rib without any motor or sensory paralysis. Operator was of the opinion that primary arterial obstruction existed at the site of the cervical rib.

"Neck was opened with a V-shaped incision, one arm of which was parallel with the posterior margin of the sternomastoid, and the other to the superior border of the clavicle. After reflecting the skin flaps the anterior scalenus muscle was identified, and divided over the subclavian artery. The subclavian artery at this site showed definite constriction with dilatation distal to the point of constriction. The operator estimated that the artery was constricted to one-half its normal size, and the dilatation distal to the obstruction made the artery approximately twice its normal size. Immediately posterior to the artery there was a mushroom-like projection of bone and the artery was incarcerated under the superior overhanging edge of a bone tubercle. Whether this bone tubercle represented a short cervical rib, or whether it was attached to the normal first rib could not be determined without what the operator considered an unnecessary additional exposure. The roots of the brachial plexus appeared quite normal. The bone tubercle was removed with Rongeur forceps. The subclavian artery was then palpated from its origin to a point well below the inferior border of the clavicle. There was good pulsation of the artery at all sites at which it could be felt. After complete release of the artery at the site of constriction, no pulsation could be felt in the radial artery. The artery was protected from contact with the denuded bone by suturing a pedicle flap of fat and areolar tissue over the raw bone surface. The subclavian artery appeared perfectly free and unobstructed throughout its entire course, and there was certainly no abnormality in the brachial plexus. The wound was closed with interrupted fine silk."

Immediately following the operation, no pulse was felt in the right brachial or radial arteries. When the patient recovered from the anesthetic, she stated that the hand and forearm "felt more natural," and there was an easily demonstrable pulsation in the axillary artery, and feeble pulsation in the brachial

and radial arteries. Forty-eight hours after operation the pulsation in the wrist was still quite feeble, but more easily felt than at twenty-four hours after operation. The patient stated that at times she noticed marked blushing of the hand, and that while the hand was red, it felt cold.

Five weeks later the necrotic end of the index finger was removed. The arm still blanched on elevation and the radial pulse could not be felt, but all subjective symptoms had been relieved. The patient was seen again in September, 1948, and she stated that the arm was "perfectly well." There was no difference in the color or warmth of the two hands. The radial pulse still could not be felt, but there was a palpable pulse in the proximal half of the brachial artery. The following conclusions were made by Dr. Brooks: (1) Local irritation of an artery without local mechanical obstruction may produce vasoconstriction of branches far beyond the site of local irritation; (2) Persistent vasoconstriction may produce anatomic arterial obstruction.

Comment

Many theories have been advanced to explain the vasospasm and arterial changes in the absence of any gross abnormality of the brachial plexus. Todd[7] showed by anatomic dissections that the lowest trunk of the brachial plexus was as a rule in closer apposition to the first thoracic rib or cervical rib than was the subclavian artery. He postulated that the vascular changes were due to paralysis or irritation of sympathetic fibers in the lower cords. Telford and Stopford[8] elaborated on Todd's views, and reported 3 cases with gangrene of fingers without any evidence of compression of the subclavian artery at operation. They demonstrated in cadavers that the sympathetic fibers to the arteries in the arm and forearm may be bunched in the lowest cord of the plexus, and, therefore, subject to irritation, resulting in distal vasoconstriction. They also fed ergot to Leghorn cockerels for eight weeks and found a proliferative endarteritis and thrombosis of the smaller arteries of the comb. The veins were not affected. Blair, Davies and McKissack[9] agree with Telford and Stopford and reported the autopsy findings in the case of a patient with cervical rib with preoperative absence of ulnar, radial and brachial pulses. They found a chronic aseptic inflammatory lesion in the nerves adjacent to the rib. This was marked by thickening of the endoneurium and proliferation of the endoneurial nuclei in the lower trunk of the plexus. However, they found no separate trunk of unmyelinated fibers as did Telford and Stopford. Eden,[10] Lewis and Pickering[11] disagree with this theory, and believe that the vascular changes are secondary to direct trauma to the artery, giving rise to thrombosis and distal emboli. Eden tabulated 45 cases of vascular complications of cervical ribs from the literature and added two of his own. Thrombosis of the arteries in the forearm persisted despite resection of the rib and relief of all symptoms. He concluded that vascular changes are due to damage to the

wall of the subclavian artery by intermittent compression between the clavicle and cervical or first thoracic rib.

CASE 3. John M., white male, aged 52, three and one-half months before admission to the hospital in December, 1947, had dropped a heavy weight on the dorsum of his right foot. X-rays were said to have revealed no fractures, and he was treated with snug dressings and a plaster cast. These were removed because of severe pain in the extremity. The foot soon became cold, bluish, sweaty and painful, and the patient was unable to bear any weight on it. Examination revealed a cold, clammy, cyanotic right foot with absent posterior tibial and faint dorsalis pedis pulses. Pulsations in the left foot were normal. Roentgenograms showed spotty decalcification of the metatarsals, diagnosed as Sudeck's atrophy.

Premliminay tests indicated marked vasospasm with temporary relief of pain (fig. 1). A right lumbar sympathectomy was performed. The foot became

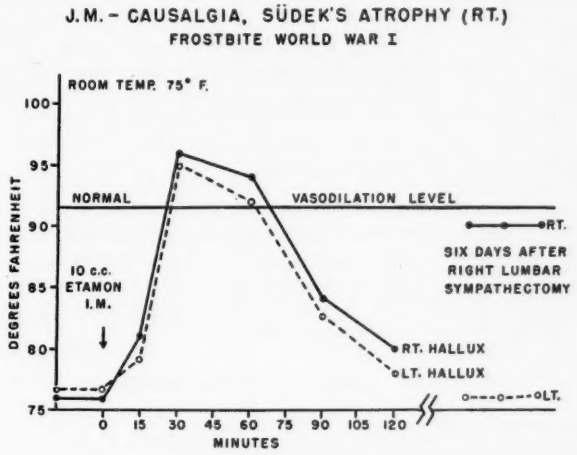

Fig. 1

warm and dry, with good pulses, and early weight bearing was instituted. Comparative x-rays three months later showed replacement of calcium, and the patient had resumed his normal life as an Alabama farmer.

COMMENT

A variety of names have been applied to this post-traumatic syndrome described by Sudeck. Miller and de Takats[12] use the term post-traumatic dystrophy of the extremities, and point out that the injury is usually mild and involves nerve trunks, vessels or periarticular structures. They advocate repeated injections of procaine into the injured area, paravertebral sympathetic block, and/or sympathetic ganglionectomy, depending on the severity of the syndrome. In the case cited above there was immediate relief of pain

and early recalcification of bone. It seems likely that many cases are not correctly diagnosed, since diffuse osteoporosis occurs in long-standing cases with the diagnosis being disuse atrophy.

CASE 4. Kermit H., white male, aged 37, in 1936 received a shotgun charge, from a distance of 20 feet, in the lateral aspect of the left upper thigh. He was hospitalized for one week and stated that there was no excessive hemorrhage. He had no further trouble until his induction into the Army in 1941. He began having pain in the left calf, with some swelling, and a high saphenous ligation with retrograde injection was carried out. He continued to have intermittent claudication, but was sent to the Pacific theatre in 1944, where he developed several ulcers on the foot and distal leg. He was returned to this country and discharged in 1945, with the ulcers healed. In April, 1948, severe pain was noted in the left foot and leg, with subjective coldness of the foot. He was treated by his family doctor with penicillin and elevation of the foot and hot applications to the foot. The pain in the foot increased until sleep was impossible. A week before admission dark areas appeared on the lateral border of the foot.

Examination revealed a moderately obese, florid male, whose blood pressure was 100/60. There was marked pigmentation of the medial aspect of the left leg, with gangrenous areas 3 cm. in diameter on the sole and lateral aspect of the foot. The foot was cold and clammy. There was a healed scar in the upper femoral region, and good pulsations of both common femorals. The peripheral pulses were absent on the left and there was slight swelling of the left leg. There were no signs of an aneurysm or arteriovenous fistula. There was rapid blanching of the foot on elevation, and oscillometric determinations were subnormal. Arteriograms showed the femoral artery to be patent from groin to knee. Skin temperature determinations revealed only slight rise on the left following one gram of tetraethylammonium chloride intramuscularly, and later 100 milligrams of procaine intraspinally (fig. 2).

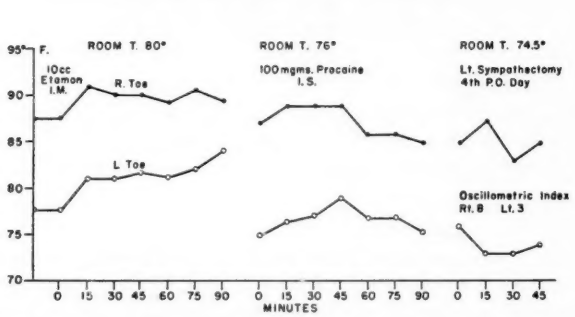

Fig. 2

A left lumbar preganglionic sympathectomy was done with little objective improvement in the foot. The ulcers failed to heal, and several weeks later amputation of the leg was performed after intermittent venous occlusion and other measures had proved unsuccessful.

Examination of the specimen revealed an ulcer 5 by 2 cm. on the posterolateral aspect of the foot. There was no thrombus in the posterior tibial artery, but microscopically there was subintimal fibrosis with reduction in the diameter of the lumen, but no endothelial proliferation. There were none of the characteristic changes found in advanced thromboangiitis obliterans.

Comment

This case was instructive because of the likely possibilities which were narrowed by arteriograms and examination of the amputated leg, as well as the advanced circulatory changes secondary to vasospasm. Since there is no evidence that this patient had femoral arterial thrombosis, arteriosclerosis or thromboangiitis obliterans, we must consider a post-traumatic disorder. Pain, coldness, cyanosis, sweating and ulceration were all present. There was also edema and absence of pulsation. Most of these signs had been present since 1941. The failure of sympathectomy to improve the leg and foot was not unexpected, but it was felt justified before resorting to amputation, since the beneficial effects of sympathectomy cannot always be accurately predicted.

Shumacker and Abramson[13] have pointed out that there is less likelihood of a successful outcome in those cases in which a long period of time has elapsed between injury and adequate treatment. This patient (and several others in our series) had had repeated trauma to the same extremity. Earlier recognition and treatment of this group of patients would yield far better results than can ever be obtained in arteriosclerosis obliterans or thromboangiitis obliterans.

Case 5. Lewis W., white male, aged 37, in 1942, while taking daily marches at a port of embarkation, had developed a sore left leg and had had two distinct pulmonary infarcts. A few days later a thrombus was removed from the left femoral vein and the vein ligated. Thrombophlebitis of the extremity followed and the patient remained in the hospital for several months before being discharged. He continued to have some pain and swelling on walking, and after several years noted some enlarged veins in the left thigh and leg. He was admitted to the Thayer General Hospital in September, 1947. The left calf and lower thigh were 2.5 cm. larger than the right at corresponding levels. The peripheral pulses were said to be palpable. A diagnosis of chronic thrombophlebitis was made, an elastic bandage was applied, and the patient discharged.

He was readmitted in July, 1948, because of increasing varicosities and "weakness of the left leg." Venograms showed both the deep and superficial system to be patent, although the lumen of the femoral vein was greatly diminished and the exact site of ligation could not be determined. It was noted that the dorsalis pedis and posterior tibial pulses were diminished as compared to the other foot. After slight exercise they became even weaker, and on several occasions were not palpable. Following 100 mg. of procaine intraspinally, the skin temperature of the toes rose from 72 to 89.5 degrees (room temperature 74

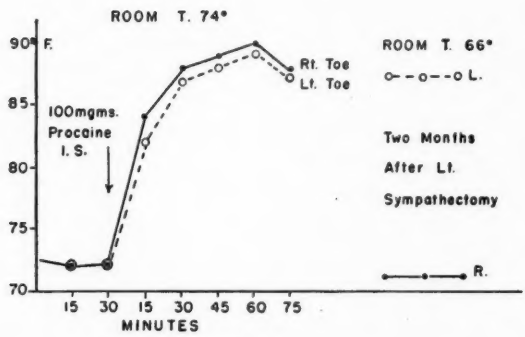

Fig. 3

degrees) (fig. 3). A clinical trial of 50 mg. of Priscol four times daily was found to increase his walking range. A preganlionic lumbar sympathectomy and an exploration of the femoral artery were then done. There was increased fibrosis and constriction of the artery at the site of previous vein ligation, and the artery was freed from all scar tissue. Postoperatively, the dorsalis pedis and posterior tibial pulses were increased and the walking range greatly increased. Skin temperature six weeks later in a room of 66 degrees with patient exposed for 45 minutes showed the right hallux to be 71 degrees, while the left remained at 87.5 degrees. On a recent checkup visit the patient stated that while his "left leg was not as good as the right," he could now "run the cows," whereas before operation he could not.

Comment

We assume that the ligation and thrombophlebitis both contributed to this patient's disability. The diminished arterial flow was picked up by alert house officer at the Veterans Hospital. It seems likely that the "unbridling" of the vessel was an important step in improving his circulation. If our assumptions are correct in this case, then at regular intervals we should check the arterial circulation in those patients having venous ligation and/or venous thrombosis.

Case 6. Sam B., white male, aged 24, in April, 1945, received a rifle wound of the left foot, the bullet entering near the medial malleolus and exiting just below and anterior to the lateral malleolus. Six operative procedures were carried out during a two year hospitalization period. On admission to the Thayer Veterans Hospital in October, 1947, there was a 2 by 3 cm. ulcer on the medial aspect of the ankle, surrounded by a 6 by 12 cm. area of scarred, discolored skin. The foot was cold, clammy and cyanotic. The posterior tibial pulse was absent and there was marked limitation of motion of the ankle.

Skin temperature determinations revealed marked vasospasm (fig. 4). A left preganglionic lumbar sympathectomy, excision of the ulcer and scar tissue, and

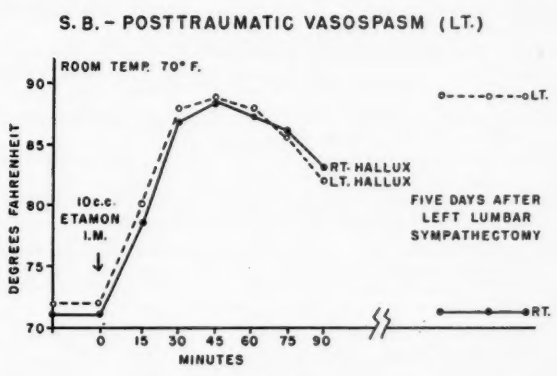

Fig. 4

application of a skin graft were done at the same time. Postoperatively, the foot became painless, warm and dry, and the graft healed well. Checkup examinations since have shown no further trouble.

Comment

Many patients with varying degrees of trauma and varying degrees of vasomotor disturbance have been encountered. Shumacker and Abramson[13] have stressed the early recognition and treatment. In most instances, repeated paravertebral blocks will be sufficient. Those patients with ulceration will likely require excision of the ulcer and grafting, with or without sympathectomy. We have felt that sympathectomy has been of real value where vasospasm was present with ulceration.

Summary

Arterial spasm in an extremity may be secondary to a variety of common causes. Persistent or recurrent vasospasm may be disabling, and, in some instances, may result in irreversible arterial changes. Early recognition and appropriate treatment give excellent results.

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DISEASES OF THE BILIARY TRACT ASSOCIATED WITH CHOLELITHIASIS*

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GALLBLADDER calculi should be removed when the diagnosis is established unless there is a definite contraindication to operation. The removal is accomplished by cholecystectomy. The opposite of this clinical rule also holds true. Cholecystectomy should not be done with a functioning gallbladder unless biliary calculi are present. Cholecystostomy is resorted to only in the patient whose general condition does not permit adequate anesthesia and the additional minutes of operating time. There is no local inflammatory process that contraindicates cholecystectomy.

When the patient harbors gallstones over a long period, damage occurs to the gallbladder, bile ducts, liver and pancreas. A review of consecutive cases revealed that there was damage to the bile ducts, liver or pancreas in 50 per cent, and these advanced cases are presented in Chart 1. The duration of symptoms varied greatly but averaged about 5 years.

The instances in which the damage was confined to the gallbladder were not abstracted. Briefly, biliary calculi damaged the gallbladder in the following ways:

The presence of the stones in the gallbladder frequently is associated with a low grade inflammatory process, chronic cholecystitis. This is due in large measure to mechanical irritation or repeated bouts of obstruction by the stone.

Obstruction of the cystic duct by the calculus produces acute cholecystitis. Heavy inflammatory reaction results. If the obstruction is relieved, the reaction may largely subside. Empyema of the gallbladder is a type of acute cholecystitis in which crystallization occurs. Less frequently actual suppuration is present.

Vascular occlusion when localized, as in the erosion of the gallbladder wall by a calculus, rarely results in a free perforation. A walled-off perforation with a pericholecystic abscess is much more common. General vascular occlusion of the blood supply results in gangrene of the gallbladder.

Since carcinoma of the gallbladder is almost always associated

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Read before the seventeenth annual Postgraduate Surgical Assembly of The Southwestern Surgical Congress, Biloxi, Miss., May 23-26, 1949.

CHART 1

Case No.	Hosp. No.	Age, Sex, Prev. Pregnancy	Symptoms Duration	Icterus Index	Cholecystogram	Operation				Result
						Stones in	Gallbladder Wall	Bile Ducts, Etc.	Procedure	
1	128, 491	64 F Yes	Dyspepsia, colic, 7 yrs.	58 69	Non-functioning gallbladder	Common duct and gallbladder	Gangrenous	Common duct filled with pus; hepatic duct sealed	Choledochostomy Cholecystectomy	Recovered (slowly)
2	126, 553	38 F Yes	Severe, 2 mo.	61	None	Common duct	Fibrotic thick wall occluded	Common duct occluded	Choledochostomy Cholecystostomy	Recovered
3	88, 679	34 F Yes	Colic, many years	34	Non-functioning gallbladder	Gallbladder	Thick wall inflamed	Common duct dilated	Choledochostomy Cholecystectomy	Recovered
4	146, 363	52 F Yes	Colic, 12 yrs.	17	Cholelithiasis	Gallbladder	Chronic inflammation	Common duct not dilated	Cholecystectomy	Recovered Episode, pain 2 wks. later. Recovered
5	113, 655	70 F Yes	Pain 20 yrs. Colic 20 yrs. Diabetic many years	N.R.	Cholelithiasis	Gallbladder	Fibrosis	Common duct thick; pancreas hard; liver enlarged	Choledochostomy Cholecystectomy	Recovered
6	138, 428	55 M	Colic 2 yrs. Angina Pectoris 1 yr.	18	Cholelithiasis	Gallbladder	Edematous, inflamed	Dilated, thick. Pancreas hard	Cholecystectomy Choledochostomy	Recovered

CHART 1 (*Continued*)

Case No.	Hosp. No.	Age, Sex, Prev. Pregnancy	Symptoms Duration	Icterus Index	Cholecystogram	Operation				Result
						Stones in	Gallbladder Wall	Bile Ducts, Etc.	Procedure	
7	119, 234	40 M	Prev. choledochostomy, chronic pancreatitis, biliary fistula, diabetes		Large gallbladder cholangiogram. Obstruction common bile duct	None	Dilated	Pancreas indurated. Common duct small, fibrotic. Biliary fistula	Cholecystojejunostomy Enteroenterostomy	Recovered
8	122, 781	28 F No	2 weeks Diabetes, recent	6	Cholelithiasis	Gallbladder	Fibrosis, hyperemia	Common duct not dilated. Pancreas hard.	Cholecystectomy	Recovered. Dyspepsia persistent
9	121, 029	44 F	Pain 10 yrs. Diabetes	13	Non-functioning gallbladder	Gallbladder	Fibrosis	Pancreas indurated	Cholecystectomy	Recovered
10	120, 095	66 F No	Jaundice 1 mo.	68 58 61 77 110	None	None	Greatly distended	Pancreas hard. Common duct dilated	Cholecystojejunostomy	Recovered
11	118, 554	32 F Yes	Colic 1 yr.	18	Cholelithiasis	Gallbladder and common duct	Distended, inflamed	Common duct dilated	Cholecystectomy Choledochostomy	Recovered

CHART 1 (*Continued*)

Case No.	Hosp. No.	Age, Sex, Prev. Pregnancy	Symptoms Duration	Icterus Index	Cholecystogram	Operation				Result
						Stones in	Gallbladder Wall	Bile Ducts, Etc.	Procedure	
12	93, 889	24 F Yes	Colic, jaundice 3 mo.	21	Cholelithiasis	Gallbladder	Imbedded in liver; adherent	Common duct small	Cholecystectomy	Recovered
13	144, 582	56 F	10 yrs.	14	Cholelithiasis	Gallbladder	Large, thickened	Not dilated	Cholecystectomy	Recovered
14	114, 396	38 F No	Pain, 6 mo.	29	Poor contraction	Gallbladder	Moderately inflamed, obstructed	Not dilated	Cholecystectomy	Recovered
15	105, 075	27 F Yes	6 mo. Cholecystectomy 9 weeks previously	23	None	Common duct (Residual)	Previously removed	Dilated	Choledochostomy. Duodenectomy and transduodenal removal of stone	Recovered (left with tube in place)
16	121, 423	40 M	1 year	35	Non-functioning gallbladder	Gallbladder	Acute inflammation: 1 cm. thick	Marked inflammation prevented exposure CBD	Cholecystectomy	Recovered
17	124, 115	66 M	Many years: 15 plus	23	None	Gallbladder	Gangrenous	Not delineated	Cholecystostomy	Recovered
18	95, 377	44 F No	Cholecystostomy 16 yrs. previously	7	Cholelithiasis	Gallbladder	Fibrosis	Slight fibrosis	Cholecystectomy	Recovered

CHART 1 (*Concluded*)

Case No.	Hosp. No.	Age, Sex, Prev. Pregnancy	Symptoms Duration	Icterus Index	Cholecystogram	Operation				Result
						Stones in	Gallbladder Wall	Bile Ducts, Etc.	Procedure	
19	127,375	70 F Yes	Cholecystostomy 33 yrs. previously	10	None	Gallbladder	Perforated with abscess	Edema	Cholecystectomy	Recovered
20	130,001	36 F Yes	3 mo.	21	Cholelithiasis	Gallbladder	Fibrosis	Common duct not dilated	Cholecystectomy	Recovered
21	119,149	56 M	7 yrs.	15	Non-functioning gallbladder	Gallbladder	Localized gangrene	Surrounding edema	Cholecystectomy	Recovered
22	128,687	62 M	8 yrs.	33	Cholelithiasis	Gallbladder	Imbedded fibrosis. Marked edema	Dilated, inflamed. Liver friable	Choledochostomy Cholecystectomy	Recovered
23	133,333	61 M	3 yrs.	24	Cholelithiasis	Gallbladder	Friable fibrosis	Edema surrounding	Cholecystectomy	Recovered
24	125,318	46 F Yes	3 yrs.	21	Non-functioning gallbladder	Gallbladder	Purulent fluid; gangrenous with perforation	Obscured by edema	Cholecystectomy	Recovered
25	150,008	60 F Yes	6 yrs.	13	Non-functioning gallbladder	Gallbladder	Purulent fluid; gangrenous with perforation	Obscured by edema	Cholecystectomy	Recovered

with calculi, the irritaton of the stones is indicated as the exciting factor.

Cholangiitis was present in cases 11 through 25 and also in most of the cases discussed later in the pancreatitis and hepatitis group. Cholangiitis was associated with common duct stone in 4 cases, with recurrent acute cholecystitis in 5 cases, with gangrene of the gallbladder in 3 cases and with perforation and pericholecystic abscess in 3 cases. The usual criterion for diagnosis was the presence of jaundice or the actual observation of a heavy inflammatory reaction of the common bile duct at operation.

Chronic pancreatitis was associated with cholelithiasis in Cases 5, 6, 8, and 9 and was present without cholelithiasis in Cases 7 and 10. Obstructive jaundice was present in 2 cases and diabetes in 4. The diabetes improved in all four. The procedure carried out was cholecystectomy with choledochostomy in 2 cases, cholecystectomy with spontaneous drainage from the cystic duct in 2 cases, and cholecystojejunostomy in 2 cases. There was a temporary diversion of the biliary flow (external) in 4, and a permanent diversion (internal) in 2.

Severe liver damage was the chief complication in Cases 1 through 4. Emergency procedures were necessary in Cases 1 and 2 because of liver damage characterized by rapidly increasing jaundice, high fever up to 106 degrees and developing coma. In each a large stone was impacted in the common bile duct. When a tube was inserted in the common hepatic duct there was no immediate biliary flow. They were characterized by a long recovery period with a slow fall in the icterus index.

Discussion

Cholecystectomy early in the course of cholelithiasis avoids serious complications and possible residual damage. If extra-biliary causes of non-visualization are eliminated, a non-functioning gallbladder on cholecystogram means obstruction of the cystic duct, in all our cases by stone, and requires cholecystectomy. Cholecystostomy is admittedly an inferior procedure and rarely has to be resorted to. However, in those few cases it has been life-saving.

Cholecystectomy is a safe procedure as far as local complications are concerned even in acute cholecystitis, even in the face of the most severe inflammatory reaction if done carefully. The gallbladder should be removed from above downwards. A good cleavage plane is present and can be entered with careful dissection. The dissection is carried only as far as can be done safely (usually just

above junction with cystic duct). If a small area of mucosa remains it can be stripped out after the removal of the gallbladder.

Removal of a functioning gallbladder should be avoided if no biliary calculi are present, primarily because the results are notoriously poor. Cholecystectomy done with an erroneous diagnosis is practically eliminated. Cholecystectomy is of no help in biliary dyskinesia and may increase the symptoms. In 50 consecutive cholecystectomies 49 had biliary calculi.

Four out of six patients with chronic pancreatitis were diabetics, and in each instance the diabetes improved markedly. The relationship is to be reported later by the medical department.[12]

Present day medical therapy seems of great value in the postoperative period in the cases of severe liver damage.

SUMMARY

1. Gallbladder calculi require cholecystectomy.

2. Removal of a functioning gallbladder is indicated only when biliary calculi are present.

3. With proper interpretation and exclusion a non-functioning gallbladder on the cholecystogram means obstruction of the cystic duct usually by calculus, and requires cholecystectomy.

4. Infection is secondary to biliary calculi.

5. When in doubt as to the advisability of a choledochostomy, do one.

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DUPLICATION OF THE CECUM (ENTERIC CYST)

Report of a Case

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BENIGN cystic lesions of the alimentary tract have been reported in the literature under various names—"enteric cysts," "enteroid cysts," "enterogenous cysts," "enterocystomas," "giant diverticuli," "inclusion cysts," "ileum duplex," and "duplications of the alimentary tract." The last mentioned title—"duplications of the alimentary tract" is the one which has been proposed by Ladd and Gross[1] as the term which is most all-inclusive and which serves best to bring this entire group of lesions together as a single entity. As defined by these authors,[2] "duplications are spherical or elongated hollow structures which possess a coat of smooth muscle, which are lined by mucous membrane, and which are intimately attached to some portion of the alimentary tube."

Several comprehensive surveys of these lesions have appeared in the literature during recent years and no attempt will be made to review the literature in this presentation. The lesions have been found in all portions of the alimentary tract, from the base of the tongue to the rectum. The largest number have been found in the terminal ileum and ileocecal region, and Drennen,[3] in 1931, was able to find 23 reported cases in this area. In 1939, Pachman[4] reported 28 cases in the ileocecal area; in 1940, Sawyer[5] reported 30 cases and added 2 more, bringing the total to 32; and in 1946, Mehn and Morrow[6] were able to find 52 cases. Most of the articles in the literature have been isolated case reports with only a few authors reporting more than a single case. Ladd and Gross, in analyzing the experience at the Boston Children's Hospital, report 18 cases of which 7 were located in the ileum and 1 in the cecum.

The generally accepted theory of origin of these lesions is that advanced by Lewis and Thyng,[7] who state that they begin as small epithelial buds which push out from the embryonic intestinal wall into the mesenchyme. At first solid, these buds may fail to regress and become vacuolated or cystic. If a communication with the main gut persists we have the formation of a diverticulum, if the bud becomes completely pinched off and no communication persists we have the anatomic prerequisite for a cystic type of duplication.

A characteristic feature of such duplications is that they are contiguous with and strongly adherent to that portion of the alimen-

tary tract from which they arise. In fact the muscular coat may be so intimately fused with the muscular coat of the bowel proper that on microscopic examination there appears to be a single common muscularis. This is important from a surgical standpoint since the lack of a line of cleavage prevents the cyst from being "peeled out" as can be done with mesenteric cysts. The cysts are lined with mucosa which is intestinal in type but which does not necessarily correspond to that at the segment of bowel removed. The contents of the cyst vary considerably and range all the way from a clear colorless fluid to a thick mucoid material.

Since the lesions are congenital the large majority of reported cases have occurred in infants and children. This is particularly true of those cysts developing in the ileum, for in this location they are apt to produce obstructive symptoms early in life. In the large bowel the onset of obstructive symptoms appears much later, and consequently, a few of these patients have reached adult life before the lesion became manifest.

The present case is unusual in that the patient was 63 years of age. Review of the literature reveals only one other case in this age group—a case reported by Quensel in a woman of 62.

Case Report

J. P. H., a 63 year old white male, was admitted to the hospital on Oct. 17, 1948, for prostatic resection. This procedure was carried out uneventfully and he made a very satisfactory recovery. During this hospitalization he mentioned rather casually to his attending physician that he was having occasional slight attacks of shifting, gaseous abdominal distress. During these periods he stated that there was occasionally some slight tendency to diarrhea. The above symptoms had been noted for only the past few months. There had been no weight loss and he had never noted any blood, pus or mucus in the stools. A barium enema was given and a suggestive filling defect was encountered in the cecum. He was advised to return for a second study of his colon after he had recovered somewhat more from his prostatic resection.

The patient returned on Feb. 16, 1949, and roentgen examination of the colon, using the barium enema, revealed a well defined filling defect in the cecum. The lesion appeared rather large, but the margins were smooth and quite regular. The cecum itself was not narrowed or deformed, and at fluoroscopic examination no significant distortion of the mucosal folds was noted. Barium could not be made to enter the terminal ileum (fig. 1a).

Following evacuation of the barium enema, air contrast studies were performed. The observations made during the barium enema study were confirmed in detail, and again the impression was gained that the filling defect lacked some of the usual characteristics of carcinoma of the cecum. However, the roentgen diagnosis was "Filling defect in the cecum, probably carcinoma" (fig. 1b).

On physical examination a smooth rounded mass could be felt in the right side of the abdomen which was about the size of a tangerine. It was located

Fig. 1a. Filling defect in the cecum as seen during barium enema study.

Fig. 1b. As visualized by air contrast technic, the lesion is partly outlined by a thin margin of barium, and in addition is demarcated by its slightly increased density as compared to the surrounding air.

slightly above the level of the umbilicus and about midway toward the flank. It was freely movable in all directions and only very slightly tender. The remainder of the abdomen was negative.

A diagnosis was made of tumor of the cecum, probably carcinoma, and he was admitted to the hospital and prepared for exploration. Operation was carried out on Feb. 24, 1949. The cecum was found to be the site of a large, smooth, rounded mass which felt cystic and gave the impression of perhaps being within the lumen of the bowel. The cecum was accordingly opened on its anterior aspect and the tumor viewed from within the bowel. It was then determined that the mass was actually within the layers of the bowel wall on the posterior aspect of the cecum. It bulged up into the lumen of the cecum carrying the ileocecal valve upward so as partially to occlude the opening. The mucosa over the mass was everywhere intact and normal in appearance. A few lymph nodes were present adjacent to the cecum and one of these was submitted to the pathologist for frozen section. It was found to consist of a waxy homogenous debris. The mesenteric lymph nodes and liver were uninvolved. It was decided that we were dealing with a benign lesion, but, because of its location and intimate relationship with the cecal wall, it was obvious that resection would be necessary. The opening in the cecum was therefore closed and the cecum was mobilized and resected together with the ascending colon, hepatic flexure and a segment of the terminal ileum. The continuity of the intestinal tract was restored by a side to side ileotransverse colostomy.

The postoperative course was smooth and uneventful and he left the hospital on the 14th postoperative day.

Pathology Report

Gross Findings: The specimen consisted of terminal ileum, cecum, and a portion of colon. Just above the ileocecal valve there was a smooth rounded

Fig. 2a. The gross specimen showing the interior of the cyst at its attachment.

tumor mass that measured 10 centimeters in diameter. It appeared to be attached to the posteromedial wall of the colon (fig. 2a). It bulged into the lumen of the colon and the mucosa in this area had been somewhat flattened out and a few areas showed mucosal hemorrhage. Upon incising the tumor it was found to contain a thick tenacious mucoid material that was gray in color.

The total quantity of this mucoid material was estimated at about 100 cubic centimeters. The inner surface of this cyst had the appearance of colon mucosa but was somewhat flat and smooth. There were no papillary outgrowths. The cyst had extended out through the muscular portion of the colon, and in one area the wall consisted only of serosa. In addition, four lymph nodes were present in the mesentery of the colon. Two of these appeared hyperplastic and two of them had undergone necrosis.

Microscopic Findings: Sections from various portions of this cyst showed it to be lined by somewhat flattened colon type mucosa. About 80 to 90 per cent of the epithelial cells showed a tendency to secrete mucus. The epithelium was completely uniform in its appearance and showed no evidence of malignant change.

At the dome of the cyst, where it projected into the colon, there were the following layers from within outward: (1) the flattened cyst mucosa with its muscularis mucosa, (2) a narrow fibrous submucosa, (3) a rather wide band of common muscularis, both circular and longitudinal, (4) the submucosa of the colon, and (5) the true mucosa of the colon itself (fig. 2b). In other parts

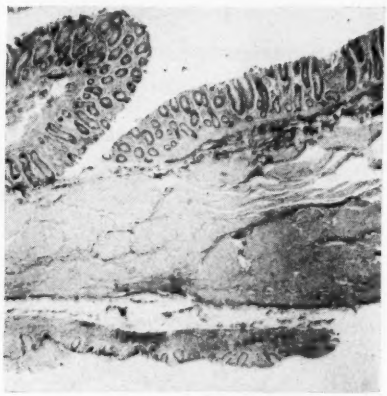

Fig. 2b. Photomicrograph of the wall of the cyst showing the common muscularis. The colon mucosa is above and the cyst lining is seen below. (Hematoxylin and Eosin, 15X)

of the cyst the bulky muscularis of the colon was very closely associated with the cyst lining except in the area where only serosa made up the wall.

Inflammatory changes were slight. A few lymphocytes and eosinophils were seen in the mucosa of the cyst and also in the mucosa of the colon proper.

The lymph nodes were completely free of any evidence of metastatic growth. Two of these had undergone necrosis with complete loss of cell detail except for the capsules. The other two lymph nodes showed simple hyperplasia.

Summary

Another case of duplication of the cecum ("enteric cyst") is added to the literature. This case is unusual because it occurred in

a patient 63 years of age who was even then experiencing only mild obstructive symptoms. The correct diagnosis was made only after the abdomen had been opened with a presumptive diagnosis of carcinoma of the cecum.

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SPINAL ANESTHESIA AND SOME OF ITS MODERN ASPECTS

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MODERN anesthesiology is demanding more exacting procedures and as a consequence is requiring more skill on the part of the administrator. Anesthesiology has maintained its stride with surgical skill and has often paved the way for the surgeon to accomplish his undertakings in a successful manner. These two fields of medicine have in the past cooperated and worked together as a team, and this has brought about remarkable accomplishments. Now that each of these branches of medicine is recognized as a highly specialized field, it is even more necessary that they cooperate, coordinate, and unite in their efforts for better advancement. It is therefore my purpose to offer a few suggestions which I hope will be helpful with the use of spinal anesthesia.

Spinal anesthesia, as it is commonly and I believe inappropriately called, is objectionable when suggested to many patients and to some surgeons. It is regrettable that such a feeling is prevalent when there is really no sound or logical basis for its existence. Rumors of spinal anesthesia spread by hearsay from one individual to another do not have the faintest resemblance to the actual facts and therefore a bad name has become attached to a good anesthetic procedure.

How are we to overcome this reputation? In the first place, let us avoid the use of the term "spinal anesthesia" and substitute one of the following: regional nerve block, lumbar nerve block, subarachnoid, or intrathecal anesthesia. These do not sound nearly as frightening as "spinal" and will give you exactly the same results. The term "lumbar block" is a good modern name for a modernized anesthetic procedure.

In the second place, the experience of lumbar nerve block can be made pleasant and desirable for the patient, simply by strict attention and adherence to a few modern aspects of technic. These are: first, a careful, positive, and easy technic of administration; second, the selection of drugs that are low in toxicity and give the longest anesthesia; third, no supplemental anesthesia with intravenous sodium pentothal to relieve psychic factors; and fourth, a better means of controlling hypotension.

Let us first discuss the aspects of technic. For example, when

Read before the seventeenth annual Postgraduate Surgical Assembly of The Southeastern Surgical Congress, Biloxi, Miss., May 24, 1949.

making a spinal puncture, the accuracy and certainty with which one introduces the needle into the subarachnoid space demands as much skill as the marksman aiming at a target. The sights of his rifle must be on a direct line with the target, from the muzzle of the gun and his eye. The trigger pull is so exacting that it does not deflect the muzzle of the gun from the time the trigger squeeze is applied until the hammer is released. The same applies to inserting the spinal needle. It is essential to use a single direct thrust of the needle, with a minimum of trauma, to introduce the point into the subarachnoid space.

The patient is placed on the table in the lateral position. The thighs and neck are flexed, forming a curve of the spinal column and spreading the dorsal spinous processes. The hips and shoulders are exactly square with each other and the dorsal spines form a straight line from the sacrum to the seventh cervical spine. A perpendicular line drawn from the crest of the ilium toward the spine will fall through the fourth lumbar interspace. For a low block anesthesia the fourth interspace is used, but for upper abdominal anesthesia the third is used. The tips of the index and middle fingers of the left hand are placed astride the selected interspace and with pressure the skin is immobilized. A wheal is made at this point, using 1 per cent novocaine with ephedrin and a 1½ inch 23 or 24 gauge needle. Following this, the space between the dorsal spines is infiltrated by introducing the needle through the wheal and injecting slowly as the needle is advanced. This makes insertion of the needle for the block free of pain or discomfort to the patient. With skill and care most patients do not realize the needle has been inserted.

When introducing the needle for the block, it is remembered that certain structures are to be traversed. The skin and subcutaneous tissues over the interspace are immobilized in the same manner as mentioned above. The needle, which is a 20 or 22 gauge 3 inch needle with stylet, is inserted through the wheal exactly in the midline. It is then advanced about five-tenths of a centimeter through the next layer which is the dorsal spinous ligament. Now the needle is anchored in the midline. The hub of the needle is held between the left thumb and the first finger, resting on the forearm on the patient. In this manner the needle is directed so that it is pointing at the center of the subarachnoid space laterally and slightly cephalad. With the thumb of the right hand on the base of the needle, anchoring the hand as a dentist holds his drill, the needle is advanced slowly. It will now pass through the intraspinous ligament into the ligamentum flavum, through which it passes with a snap. As the needle passes on, it penetrates the paradural space.

Pressing the needle further, there is another quick snap with the feeling of a lack of resistance. The needle point is now in the subarachnoid space having passed through the dura and the arachnoid which lies close to the dura on the inner side. When the stylet of the needle is removed at this time, the spinal fluid will flow freely. Care must be taken not to strike the anterior canal wall with the point of the needle because it will result in hemorrhage and may cause the anesthesia to be incomplete or to fail in its desired results.

Secondly, let us consider the selection of drugs that are low in toxicity and give the longest anesthesia. There are several drugs which can be used to produce the block anesthesia, and of these, novocaine or procaine is the oldest, least toxic, and has the shortest duration. This one is preferred for continuous block anesthesia by fractional dosage. Pontocaine nymphanoid, or crystalin pontocaine, used as a hypobarric or hyperbarric solution is preferred for the single block anesthesia. The hypobarric solution is prepared by dissolving 10 mg. of the crystals in 5 c.c. of distilled water, while the hyperbarric solution is prepared by dissolving the crystals in a 10 per cent solution of dextrose, using 1 c.c. to each 10 mg. Pontocaine will give about one-third longer anesthesia than procaine, using about one-tenth the equivalent dose. Other drugs are mentioned in passing, such as metycaine and nupercaine, but these are seldom used because of their increased toxicity.

To enhance further action, a vasopressor drug such as ephedrine sulphate or adrenalin can be added to the solution. By using one of these drugs, one can prolong the action of the anesthesia as much as 20 to 30 per cent. The addition of adrenalin has been proved to be more effective than the addition of ephedrine according to Adriani. With the addition of the vasopressor and the use of the hyperbarric solution, the total dose of the drug used for lumbar block anesthesia has been reduced to a minimum. It is no longer necessary to use the maximum dose of these drugs and consequently the toxic reactions to the drugs have been greatly reduced. The level of anesthesia is more easily controlled by the use of either the heavy or the light solution.

When pontocaine is used, the maximum dose of 10 mg. is seldom exceeded, while for perineal anesthesia, such as saddle block for hemorrhoidectomy, 3 or 4 mg. are given. The duration of anesthesia should be from one and a half to two hours. If the operation is to be definitely over two hours, we prefer to use the continuous fractional technic as introduced by Touhy, for again we can keep the fractional and total dose to a minimum and thus avoid toxic reactions.

In the third instance, with the combination of anesthetic procedures and technics first advanced by Lundy, modern anesthesia has become a much more pleasant experience than formerly when it was a dreaded ordeal of thinking one might suffocate or that one might never awaken from the induced sleep. Most people want to be unaware of their surroundings in the operating room, others do not want to be put to sleep, and no one wants to be hurt or to suffer discomfort. Therefore, by combining procedures, we can strike a happy medium.

It has become almost a routine with us to use the combined procedures. The block anesthesia is induced and when the desired level of anesthesia is established, immediately a needle is introduced into a vein in the arm and sodium pentothal is administered fractionally. A level of hypnosis is maintained so that the patient is unaware of his surroundings. This simply means hypnosis to the point of sleep from which he can be aroused or from which he will recover in a very few minutes.

Another advantage is that should the block anesthesia not be complete, or the operator's manipulations extend beyond the field of the regional anesthesia, then the general anesthesia can be quickly induced by the pentothal and the patient will not be aware of any discomfort. The time limit of the operation may exceed the duration of the block anesthesia and again the general can be supplemented in order to complete the surgical procedure satisfactorily.

The results from the use of the combined procedures are that the anesthetic has been a pleasant experience for the patient. For example, he will not remember much more than that he came to the operating room, was placed on the operating table and then awakened to learn that the operation had been completed.

Lastly, the control of hypotension which may follow the use of lumbar block anesthesia is not so difficult as it was formerly. Now we try to prevent its occurrence by administering a minimal dose of the drug which is therefore less toxic. It is unwise to move the patient about on the table after the drug has been injected into the subarachnoid space because this will produce a prompt drop in blood pressure due to disruption of the hydrostatics of the circulation. It will also cause the anesthesia to ascend to an undesired higher level.

Patients who do not receive the pentothal sodium are more apt to have a fall of blood pressure than those who do receive it. I believe the reason for this difference is psychic, or emotional, and during this time excessive adrenalin is liberated into the blood stream. The effect of the adrenalin is an initial rise of the blood pressure followed by a loss of this action and then a depression.

It has been observed that when the tidal exchange is limited, particularly in upper abdominal anesthesia, it will be helpful to give oxygen inhalation in high concentration. The prevention of hypoxia will prevent depression and hypotension. The fluid volume is maintained by starting intravenous dextrose, normal saline, or a combination of the two as soon as possible after administering the anesthetic block.

The blood pressure must never be allowed to stay at an abnormally low level for longer than a few minutes because irreparable damage may occur as a result of stagnant anoxia.

Therefore, should all the above procedures fail to maintain a safe level of blood pressure, the vasopressor drugs can be used. I would like here to condemn the use of adrenalin for the purpose of overcoming hypotension because it is vigorous in action, does not sustain the blood pressure, and also each additional dose is less effective than the previous. One of the vasopressor drugs which is more effective is ephedrine sulphate, given intravenously, only in small doses of 15 to 25 mg. with repeated doses after five or ten minutes if necessary. Neosynephrin, which is another effective drug, can be given in 5 to 10 mg. doses. Other effective drugs are methedrin and onethyl, also used in small repeated doses.

Sometimes it is necessary to resort to blood or plasma when the proper response is not immediately obtained. "Better start blood early than wait too late." It is always necessary to use blood when true shock is present in preference to a vasopressor drug.

Since we have available more modern methods for the control of hypotension in lumbar block anesthesia there are now fewer contraindications to its use.

In summarizing, I would like to reiterate, the modern aspects of lumbar block, intrathecal, or subarachnoid anesthesia. We should do our utmost to preserve an invaluable procedure by overcoming all of the hearsay talk against its use. It behooves the administrator to improve his technic of administration because of lumbar block anesthesia is not a trivial procedure that can be executed successfully by the novice or a technician. Strict attention must be given to the selection of a drug to use. This selection refers particularly to the toxicity and size of the dose that will give satisfactory anesthesia. The combined procedure of lumbar block with sodium pentothal, for hypnosis or supplementation, is more satisfactory than either procedure alone. There should be diligence at all times, taking care that the blood pressure does not fall below safe limits, or at least not allowed to remain low for more than a few minutes.

I hope that the suggestions presented in this discussion will be helpful to you in the use of lumbar block anesthesia.

CHANGES IN EXTRACELLULAR FLUID AND CARDIAC OUTPUT IN HEMORRHAGIC SHOCK*

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THE theory of plasma loss in shock is based on the assumption that there is an excessive loss to the tissue spaces due to an increase in capillary permeability.[1,2] If this assumption were correct, then there should be an increase in the interstitial fluid in shock (and plasma would be the ideal replacement therapy). Demonstration of the inferiority of plasma when compared to whole blood in the treatment of shock in the last war has stimulated further investigations of this theory.[3,4] Determinations of changes in blood volume and thiocynate space in dogs with hypotension and hemorrhagic shock in our laboratory have shown either no significant changes or a decrease in the interstitial fluid in most instances. This, combined with similar results obtained by others,[5-11] suggests an explanation of why plasma has not proved to be the ideal replacement therapy in shock.

Procedure I: Ten mongrel dogs were anesthetized with barbital anesthesia, 250 mg/Kg. The blood volume and thiocynate space were estimated, using the methods of Gregersen.[12] Cannulae were inserted into each femoral artery and one of the femoral veins. The cannulae, containers and tubing were clean but not sterile. Blood was removed from one arterial cannula until the arterial pressure was 50 mm. of mercury. This pressure was maintained for ninety minutes and the blood volume and thiocynate space were again estimated. The animals were then sacrificed and specimens taken of the liver, spleen, intestines and adrenals, the pathology of which will be included in a later report. The interstitial fluid was calculated from the plasma volume and thiocynate space. There was an increase in the interstitial fluid in three and a decrease in seven (Table 1). The average temperature was 27° Centigrade but variations of 2° to 5° were sometimes present for a few minutes during the experiment. Cardiac outputs were determined by the pressure pulse method of Hamilton and Remington[14] and total resistance calculated by the use of the formula:[15]

$$TPR = \frac{PM \times 1332}{C.O./sec.} = \frac{\text{dynes. sec.}}{cm5}, \text{ and is expressed in absolute units.}$$

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Procedure II: Three mongrel dogs were prepared as in Procedure I and blood removed until the arterial pressure was 30 mm. of mercury. This pressure was maintained by additional bleeding or small transfusions for three hours. The blood volume and thiocynate space were then again determined. There was an increase in the interstitial fluid in one and a decrease in two animals.

Procedure III: Twelve mongrel dogs of average size (Table 3) were anesthetized with barbital anesthesia 250 mg/Kg., and bled under conditions described under Procedure I until the arterial pressure was 30 mm. of mercury, and maintained at this pressure by additional bleeding and transfusions until the pressure could not be maintained at 30 mm. of mercury without repeated small transfusions. Once this uptake of blood begins it is steadily progressive until the animal expires. Twenty-five c.c. of blood was chosen as the size of the transfusion and two transfusions were chosen as the number to demonstrate the beginning of this uptake of blood. The remaining blood was then reinfused and the blood volume and thiocynate space immediately determined. The animals were either sacrificed or placed in cages with water and dog biscuit and checked for survivals. Eight were sacrificed and four died within twelve hours. In a previous series there were 28 deaths and four survivors, using this procedure.[13] The interstitial fluid was decreased in eleven and increased in one. Cardiac outputs and peripheral resistance were determined by the methods previously described.

Procedure IV: Procedure III was performed on seven dogs except that pentothal sodium anesthesia and sterile technic were used. There were three survivors and four deaths. The interstitial fluid (Table 4) was decreased in five and increased in two.

Discussion

From these experiences with hypotension and severe hemorrhagic shock in which there was a decrease of interstitial fluid in 25 and an increase in 7, it would appear that there is usually not a loss of plasma to the tissue spaces, but a shift of tissue fluid into the blood under these conditions. The wide variation of blood removed (16 to 43 per cent) to produce hypotension, the variation (23 to 52 per cent) of blood loss to reduce the arterial pressure to 30 mm. of mercury, and the variation (33 to 64 per cent) of blood loss necessary to produce the signs of irreversibility precludes their use in evaluating the depth of shock.

The variation of blood loss per Kilogram of body weight (2.1 per cent to 5.3 per cent) to produce shock and the variation in time

necessary to produce deep shock also preclude them as good criteria of shock depth.

From these variations and similar experiences by others, it is not surprising that conflicting reports on shock continue to appear. Despite one's best efforts to maintain a controlled experiment, there are so many variables that large series by several observers are necessary to establish any one point.

All of the animals showed some swelling of their wounds that was more marked in those in which clean but not sterile technic was used. For this reason we feel that the type of shock produced was similar to the usual type encountered clinically, hemorrhage and trauma, from accidental injury.

An analysis of pressure pulse contours, cardiac output and calculated total peripheral resistance during the hypotensive period and immediately after transfusion indicates that it is impossible to differentiate the dog in irreversible shock from one that will live on the restoration of blood volume by these criteria.

Summary and Conclusions

The changes in blood volumes and thiocynate space have been investigated in dogs with hypotension and hemorrhagic shock. A decrease in interstitial fluid was found in twenty-five and an increase in seven. This suggests that there is usually not a loss of plasma to the tissue fluid space under these conditions.

These experiments combined with similar experiences of others suggest an explanation of why plasma is not as effective as whole blood in shock from hemorrhage.

The wide variation in time and the amount of blood loss before the signs of deep shock appear demonstrates the difficulties of using these criteria in establishing adequate controls.

An analysis of pressure pulse contours, cardiac output and calculated total peripheral resistance during the hypotensive period and immediately after transfusion indicates that it is impossible to differentiate the dog in irreversible shock from one that will live on the restoration of blood volume by these criteria.

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BONE DISEASES WITH OSTEOPOROTIC OR MALACIC CHANGES

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EVIDENCE of demineralization of various parts of the skeleton may be attributed to a multiplicity of causes and can be separated into a variety of entities.

Bone formation and bone resorption occur continuously and simultaneously in the normal physiology of the skeletal structure. Evidence of increased porosity may be either because: I—Bone formation is too little (osteoporosis or osteomalacia), or because: II—bone resorption is too great (hyperparathyroidism). In addition, III—a number of diseases lead to demineralization or to destruction of bone, such as metastatic carcinoma, multiple myeloma, etc.

A review of the literature indicates that much of the concept of "osteoporosis" and "osteomalacia" is based on the clinical history, x-ray findings and some clinical laboratory studies. More recently concentrated efforts have been made to determine etiology based on the physiology of bone and those things which directly affect it.

In the pathologic physiology of osteoporosis, the osteoblasts lay down an insufficient amount of bone matrix. The matrix, however, is normally calcified and, as is to be expected, normal serum and calcium levels obtain. In osteomalacia a calcium or phosphorus metabolism disorder is found leading to a lack in deposition of the complex calcium-phosphate-carbonate salt in the osteoid structure. Such a state leads to increased stress and strain with further production of osteoblastic tissue and, therefore, a high serum alkaline phosphatase level; usually a low serum phosphorus and a normal serum calcium level. In hyperparathyroidism or chronic acidosis with increased bone resorption there is a decrease in bone tissue as a whole. The bone structure weakens, which increases the activity of the osteoblasts with a resultant high phosphatase level. In hyperparathyroidism with bone resorption a low serum phosphorus and high serum calcium are usually found. In metastatic malignancy involving bone and in multiple myeloma there may be a high serum calcium, possibly due to dissolving bone salts accumulating more rapidly than the kidney can excrete them (Albright). The serum phosphorus is usually normal, though in multiple myeloma it may

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Read before The Southwestern Surgical Congress, Houston, Texas, Sept. 27, 1949.

be low. The phosphatase level may be elevated (rarely in multiple myeloma) due to increased osteoblastic activity attempting repair. Boeck's sarcoid with bone changes may show hypercalcemia, high serum phosphatase but not a high phosphorus blood level. These changes are probably due to a disordered blood chemistry and not primarily to the disease.

I. Osteoporosity Due to Diminished Bone Formation

Osteoporosis may be defined as a disturbance of bone tissue metabolism in which there is a defect either in the osteoblasts or in the bone matrix. In certain diseases (acromegaly) the cause of the osteoporosis is not entirely certain. There is usually a normal serum calcium. The phosphorus is frequently at the upper limits of normal and the serum alkaline phosphatase is normal. Certain examples of abnormal serum calcium levels have been pointed out by Albright and Reifenstein in young children or persons with excessive bone formation (Paget's disease) who have suddenly been immobilized. Under these conditions the serum calcium is elevated due to the liberation of calcium from the immobile portion of the skeleton undergoing disuse atrophy.

Osteoporosis due to a systemic cause usually affects the vertebrae, pelvis and skull and rarely involves the extremities. Osteoporosis from disuse is localized to the affected portions of the skeleton.

Osteoblastic activity can be influenced by steroid hormones, mechanical stresses and strains and certain nitrogenous substances. In osteoporosis of old age of either sex, of the post-menopausal state and in Cushing's syndrome the steroid hormones are out of balance. The steroid hormones involved in stimulating obteoblasts are (Albright): (a) adrenal cortical "N" hormone, (b) estrogen and (c) testosterone. There is one steroid hormone which inhibits the osteoblasts, the adrenal cortical "S" hormone.

The explanation for the stress and strain factor in bone formation is predicated upon the thesis that the skeleton adds to itself in order to withstand stresses and that osteoblastic activity is spontaneously activated when the skeleton is unstable.

In osteoporosis frequently associated with starvation and malnutrition, serum albumen is cited as a possible important nitrogenous precursor link which is depleted and adversely affects the production of bone matrix.

Conditions Associated with Osteoporosis

Disuse Atrophy: Disuse is a part of the stress and strain factor in osteoporosis. The skeleton adds to itself in order to withstand

stresses. This stimulus is removed in the fixation or non-use of the part, for whatever reason, and gives rise to decreased osteoblastic activity (fig. 1). Over-fixation of the part and many diseases, in-

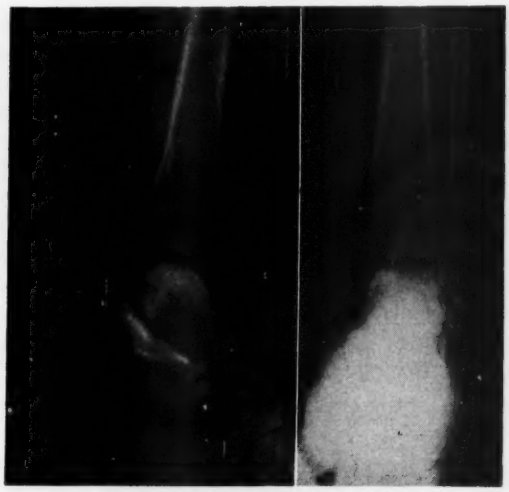

Fig. 1. Roentgenogram showing disuse atrophy in the lower tibia and fibula and bones of the foot. Note the porosity of the bones, giving the appearance of rarefaction and lack of mineralization.

cluding various forms of arthritis, such as Marie-Strumpell arthritis and Bechterew's disease, ochronosis with osteoporosis and paraphlegia may produce disuse atrophy.

The atrophy of disuse may complicate other forms of osteoporosis, as by the fixation of a fracture due to post-menopausal or senile osteoporosis. This increases the osteoporosis inviting other fractures or non-healing of the existing fracture.

In those instances where poliomyelitis, traumatic paraphlegia or sudden immobilization of the skeleton due to injury occur, an acute form of atrophy of disuse may be found. Bone formation suddenly stops, bone resorption continues, the patient developing hypercalcemia with a resultant hypercalcuria. Kidney stones are a frequent complication. If osteoporosis develops gradually the discrepancy between bone formation and bone destruction is not marked and is reffected to a minimal extent in the blood and urine calcium levels.

The treatment of the atrophy of disuse calls for high protein diet, mobilization of the affected skeleton as soon as possible, massage and, in some cases, estrogenic or testosterone therapy. If found complicating some other disease, such as Marie-Strumpell arthritis,

improvement of the primary disease may lead to skeletal mobilization, increased bone production and dissipation of the discrepancy between bone resorption and osteoblastic activity with new bone formation.

Senile Osteoporosis is characterized by weakness, easy fatigue, nervousness, dull ache in the spine with, frequently, sudden onset of pain, usually in the lower part of the thoracic region or in the lumbar region of elderly people. The x-ray studies reveal that the majority of changes are in the vertebrae. There is frequently marked decalcification with compression of vertebral bodies in their anterior portions. Non-use plays a role in the development of senile osteoporosis, which may be also partly attributable to the loss of both gonadal hormones and the cortico-adrenal, osteoblastic stimulating hormone (Albright). Space does not permit the listing of many of the commonly proposed etiologic factors which seem to us somewhat inadequate. The treatment of senile osteoporosis is essentially that of post-menopausal osteoporosis (see below).

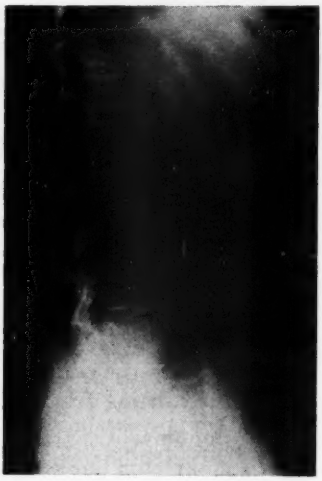

Fig. 2. Roentgenogram depicting the rarefaction and lack of mineralization of the vertebrae, associated with post-menopausal osteoporosis. The pelvis also showed similar changes in this case.

Post-menopausal Osteoporosis: The most common form of osteoporosis occurs in women after the menopause, though occasionally similar lesions are noted in the male. The average time of onset is from 5 to 9 years after an uncomplicated physiologic menopause. The osteoporosis has a predilection for involving the spine and the pelvis; the long bones are less frequently affected. The skull is almost never involved, which is an important differential

point in distinguishing this disease from osteitis fibrosa generalisata. Roentgenograms reveal osteoporosis of the affected bones (fig. 2). Collapse of some vertebrae may be present with anterior wedging of others.

Symptoms include a gradual onset of back pain, increasing dorsal kyphosis and depletion of vitality. The onset may be abrupt following injury or "strain" and the pains may be of an acute nature. The patient may evidence a nervous tension state. The fact that beneficial effects are obtained in this condition from the retention of calcium by estrogen therapy is an important argument for an etiologic hormone factor in post-menopausal osteoporosis. Gardner and Pfeiffer in extensive studies have pointed out the effects of gonadal hormones in the skeleton of animals. Estrogen stimulates cortical and trabeculated bone growth in pigeons and mice. Testosterone stimulates the bone growth in pigeons but inhibits growth in mice.

Treatment of post-menopausal osteoporosis with estrogens alone, or in combination with testosterone, has relieved skeletal pain within a few weeks or months. The patient shows an improvement in well-being and a gain in weight. The roentgen findings in general show no change, although in a few cases some recalcification takes place.

Malnutrition Osteoporosis: Osteoporosis is a common finding in states of starvation and malnutrition. These is considerable evidence to indicate that certain nitrogenous elements are necessary in the production of the bone matrix. Serum albumen is felt to be such a nitrogenous precursor, and a deficiency of this substance is found in starvation and malnutrition where osteoporosis is present. A deficiency in protein is undoubtedly responsible for some osteoporoses which have been attributed previously to a lack of calcium and phosphorus. Diet may also play a role in osteoporosis by an insufficient intake of calcium and phosphorus causing an acceleration in the resorption of bone. A diet high in calcium and phosphorus frequently will prevent decalcification, even in hyperparathyroidism.

Osteoporosis in Scurvy: Scurvy is a deficiency disease due to a lack of water-soluble vitamin C. The infantile variety is more acute in onset than scurvy found in adults. The essential pathologic feature is the inability of mesenchymal derivatives to form collagen-like cement substances in the absence of ascorbic acid. In children the roentgenogram reveals an irregular, broadened epiphyseal line with increased calcification behind the epiphyseal line, known as the "white line." The cortex of the shaft is thinned and to some extent rarefied (fig. 3). The serum alkaline phosphatase is lowered, indicating decreased osteoblastic activity.

Hemorrhage beneath the periosteum of bones and about gums is

Fig. 3. Radiographic findings in the bones of the lower extremity in a patient suffering from scurvy. Note the broadened, irregular epiphyseal lines with increase in calcification just behind the epiphyseal lines. Cortex is somewhat thinned and rarefied.

common. Motion is extremely painful and the extremities are frequently held rigid to prevent motion.

Ascorbic acid is necessary for the formation of collagenous material of all fibrous tissue structures and all non-epithelial cement substance, including vascular epithelium. A deficiency of ascorbic acid, to a less degree than in scurvy, may well account for cases of osteoporosis associated with malnutrition and hyperthyroidism.

Osteoporosis of Hyperthyroidism presents the radiographic picture of loss of density with thinning and pitting of the cortex and accentuation of the trabeculae (fig. 4). The changes are generalized but appear earliest in the smoll flat bones and the spine. Brailsford reports the appearance of an Erlenmeyer flask deformity of the lower ends of the femurs in some cases. Pain is an outstanding symptom associated with the bone changes. The serum calcium and phosphorus are normal but the calcium excretion may be considerably elevated. The state of hyperthyroidism has usually been

Fig. 4. Radiographic picture showing changes in the bone of a patient suffering from hyperthyroidism over a period of years. Note the thinning and rarefaction of the cortex, together with the accentuation of the trabeculae. (Case of Dr. I. Snapper, reprinted from "Medical Clinics on Bone Diseases" by Dr. I. Snapper; Fig. D, Plate II Interscience Publishers, Inc., New York, N. Y., 1949)

present for four or five years before the bone changes become evident. The part played by thyrotoxicosis in the etiology of osteoporosis is not certain. The prolonged use of protein for energy may result in a protein deficiency preventing the normal formation of osteoblasts or matrix. Vitamin C may be deficient or the disease may be complicated by a steroid hormone deficiency. Treatment directed towards improvement of the hyperthyroidism favorably affects the osteoporosis. The disease is not common in children.

Post-traumatic Painful Osteoporosis is characterized by partial loss of function of the affected part, varying degrees of vasomotor and trophic changes and spotty demineralization of the skeleton in the region of the trauma. Disturbances of function and aching pain are out of proportion to the trauma inflicted. The disease may be divided into three groups:

1. Painful osteoporosis with slight trauma to periarticular regions, coming on gradually over a four week period.

2. Painful osteoporosis which follows injury to the articular or periarticular tissues, usually with a fracture of one or more bones in the extremity.

3. Painful osteoporosis which follows moderate trauma to soft parts about the joints.

The mottling of the bone as seen in the roentgenogram is usually more marked in the carpal and tarsal bones and the heads of the metacarpal and metatarsal bones; the cortex is thinned and the lamellae become indistinct. The absorption of bone seems to spread; the limits of bones see mto be lost (fig. 5); ultimately, repair sets

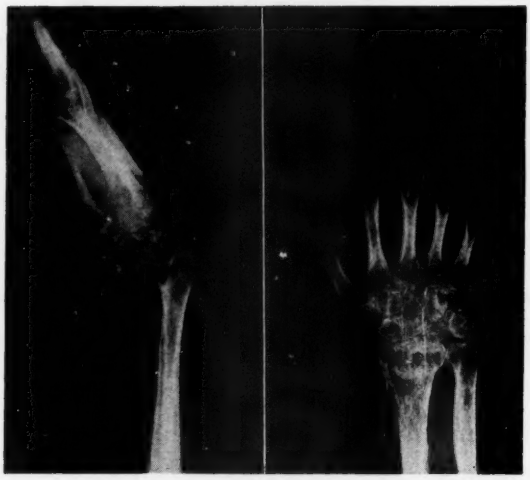

Fig. 5. Radiographic picture of bone changes at the wrist in a patient with post-traumatic painful osteoporosis. Note the excessive mottling of the carpal bones, and similar changes in the heads of the metacarpal bones and the lower end of the radius and ulna. Some of the carpal bones show wiping out of contour.

in and symptoms clear up. Roentgenograms made much later, however, show some evidence of osteoporosis. Periarterial sympathectomy in the hands of Herrman has done most to relieve symptoms and shorten the course of the disease, especially when performed in the acute phase of the malady.

Kummell's disease is an osteoporosis associated with a symptom complex which follows trauma either directly or indirectly to the buttox, head or shoulders while the spine is erect. A linear fracture of a vertebra occurs and frequently is overlooked or cannot be seen in the x-ray. Osteoporosis appears in the involved vertebra, which may ultimately lead to spontaneous vertebral collapse with localized pain, kyphosis and gibbous formation, with or without cord disturbance.

Cushing's Syndrome Osteoporosis. The pathologic condition of bone associated with Cushing's disease has many of the clinical characteristics of post-menopausal osteoporosis. One of the immediate causes of this disease seems to be an overproduction of the

Fig. 6-A

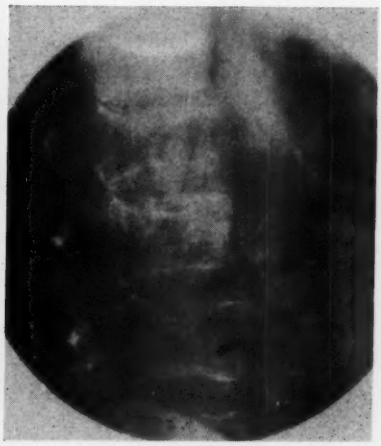

Fig. 6-B

Fig. 6-A. Osteoporosis associated with Cushing's syndrome. Note the osteoporosis present. In the vertebrae, the discs are somewhat compressed together, indicating long duration of the disease, with treatment.

Fig. 6-B. Osteoporosis associated with Cushing's syndrome. Note the osteoporosis present. In the vertebrae, the discs are somewhat compressed together, indicating long duration of the disease, with treatment.

Fig. 6-C

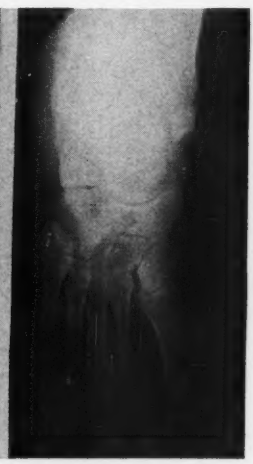

Fig. 6-D

Fig. 6-C. Osteoporosis with rarefaction of the cortex and accentuation of the trabeculae in same patient.

Fig. 6-D. Normal foot bones for comparison.

adrenal cortical "S" hormone which also accounts for the osteoporotic changes. Albright feels that the important factor in the causation of the osteoporosis is the negative nitrogen balance due to the "S" hormone, an anti-anabolic hormone, rather than to a catabolic hormone. For this reason, he feels that testosterone therapy as an anabolic stimulator is effective in treating Cushing's syndrome. The osteoporosis found in the skeleton may be minimal or extreme, with bending deformities and compression fractures of the vertebral bodies (fig. 6 A, B, C. D). In children there is cessation of skeletal growth. The skull shows osteoporosis. The skin is thin and reddened, subject to striae formation and easily bruised. Hirsutism and a peculiar distribution of fatty deposits are present. The size of the sella turcica rarely shows any alteration, though a basophilic adenoma of the pituitary gland is constantly observed associated with the disease.

Testosterone propionate has a beneficial effect on the nitrogen, calcium, and phosphorus balances and an attendant rise in the alkaline phosphatase level in patients with Cushing's syndrome. Methyl testosterone has a similar effect. The improvement seems to be brought about by the anabolic stimulation of bone growth. In some instances estrogen therapy has been of benefit, apparently through favorably affecting the calcium balance. At times a combination of estrogen and androgen therapy show increased benefit.

Acromegaly. The osteoporosis of acromegaly is due to an increase of a corticotropic pituitary hormone and to a secondary lack of gonadal hormone. There is enlargement of the feet, hands and skull (fig. 7A); the metacarpal bones and the phalanges broaden (fig. 7B); the long bones are increased in width and present coarse, heavy trabeculations. This disease does not start until after growth of the skeleton has matured, further growth of the epiphyses being impossible. The clinical abnormalities are those due to excessive periosteal bone formation with restricted new endochondral bone formation. Many patients with acromegaly show demineralization of their skeleton. There is an absence of high serum phosphatase level, an elevation of the serum inorganic phosphorus level and the absence of a hypercalcemia. The factors concerned in the osteoporosis of acromegaly may be a parathyroid adenoma, which commonly does accompany eosinophilic tumors of the pituitary gland; excessive adrenal cortical "S" hormone, the result of adrenal secretion stimulated by pituitary eosinophilic tumor (corticotropic hormone); or to a lack of some important nitrogenous element. The latter is suggested by necessity of higher nitrogen intake in acromegaly to keep the patient in nitrogen balance and preventing a subsequent loss of calcium. Hypogonadism which accompanies most cases of

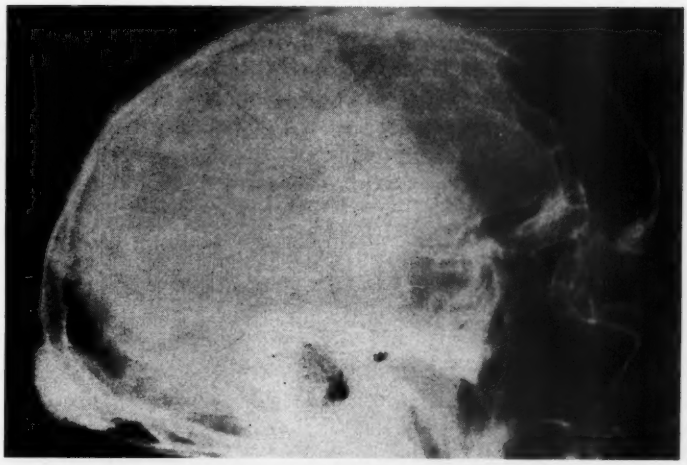

Fig. 7-A. Note the skull in acromegaly reveals enlarged sella turcica, frontal and other paranasal sinuses. The bone shows heavy trabeculation.

Fig. 7-B. Note the terminal phalanges are broadened in acromegaly. The cancellous spaces are enlarged, and in negative nitrogen balance, the bones show an increased osteoporosis.

acromegaly may well be responsible for the osteoporosis of acromegaly. The fact that the bone porosity is favorably affected by estrogen is highly suggestive, though some patients show no evidence of hypogonadism. In men who do not show evidence of gonad deficiency this explanation runs into difficulty.

Osteogenesis Imperfecta is most frequent between the ages of

8 and 16 years. It is an hereditary disease and probably involves all of the blastemal tissue giving rise to bone elements. The disturbance in the bone is much like that of osteoporosis, except that there is an apparent failure of the osteoblasts to form a normal amount of extracellular substance. The bones are fragile with accompanying fractures and deformities. In the roentgenograms the bones have a thin cortex, except where fractures have stimulated increased ossification. The shafts of the long bones are slim with broad metaphyses, sometimes foamy in appearance (fig. 8).

Fig. 8. Roentgenographically, the long bones in osteogenesis imperfecta are slim with broad metaphyses, sometimes foamy in appearance. The cortex is thinned.

Disturbances in other tissues are also present. The sclerae are blue; the structure of blood vessels, fascia, etc., seems more elastic or pliable. The condition is a dominant characteristic and is passed from generation to generation. The disease varies in severity and, if the patient survives, there is improvement when adult life is reached, at which time osteosclerotic changes may appear.

The serum calcium and phosphorus in uncomplicated cases is normal; no calciuria or phosphaturia is present. Occasionally the phosphatase may be slightly elevated.

Conditions Associated with Osteomalacia

Osteomalacia is a disturbance of bone metabolism in which calcium salts fail to be deposited promptly in the newly formed osteoid tissue. The disorder seems to be due to an abnormality in the body fluids bringing about an abnormal ratio between calcium and phosphorus, preventing the normal precipitation of the appropriate phosphate salt in the bone matrix. The body fluids either contain too little calcium or too little inorganic phosphorus to maintain a proper ratio between the two salts. An additnonal factor suggested by Albright is the insulating quality of osteoid tissue matrix. There is intriguing evidence (Albright) that osteomalacia in this country may be divided into four degrees: (1) Chemical osteomalacia with normal phosphatase; (2) Chemical osteomalacia with high phosphatase; (3) Milkman's syndrome and (4) Advanced osteomalacia. The first two degrees are not within the range of visible demineralization. The third degree (Milkman's syndrome), and the fourth degree represented by fetal, infantile, late and adult rickets show typical bone changes.

The etiologies of osteomalacia met in the United States, according to Albright, are:

A. Vitamin D lack.
   1. Simple lack of vitamin D.
   2. Resistance to vitamin D.
   3. Steatorrhea.

B. Renal acidosis.
   1. Tubular insufficiency without glomerular insufficiency.
   2. Fanconi syndrome.

C. Idiopathic hypercalcuria.

D. Hyperparathyroidism with osteitis fibrosa generalisata during transitional stage following removal of parathyroid tumor.

Milkman's Syndrome: Milkman in 1930 reported a patient with multiple spontaneous symmetrical fractures involving both the flat and pipe bones. Subsequent cases have been reported and may be found described under various designations as: (a) Pseudofractures; (b) Multiple spontaneous idiopathic symmetrical fractures, etc. The syndrome is characterized clinically by progressive difficulty in walking and pain in the lower back and extremities; radio-

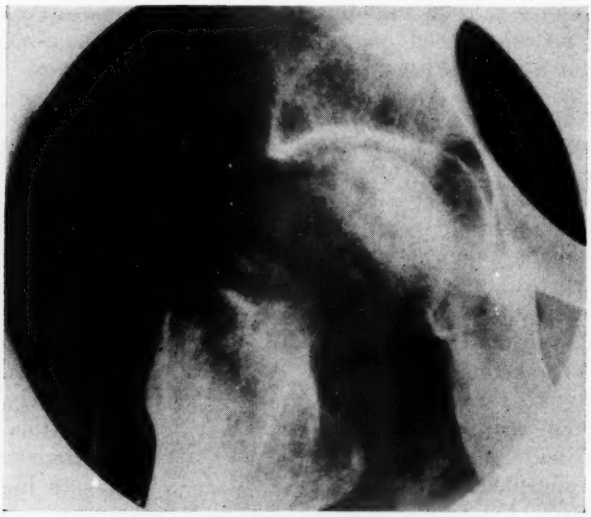

Fig. 9-A. Note the ribbon-like zones of decalcification in the neck of the femur, and in the third rib. The remainder of the bone appears normal. (Case of Dr. Irving Brick.)

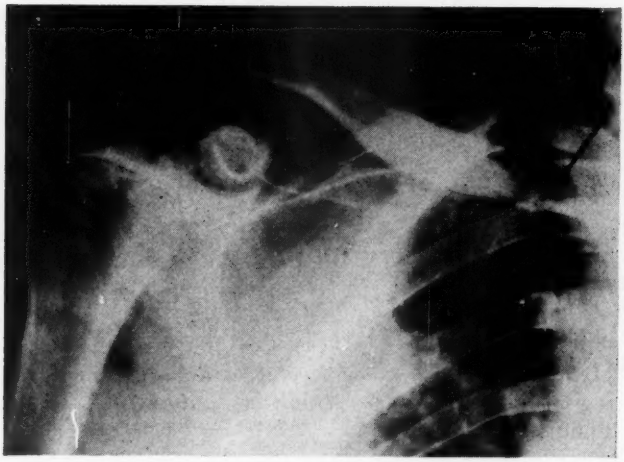

Fig. 9-B. Note the ribbon-like zones of decalcification in the neck of the femur, and in the third rib. The remainder of the bone appears normal. (Case of Dr. Irving Brick.)

logically, by ribbon-like zones of decalcification in what otherwise appears to be normal bone (fig. 9 A, B). The bone defects are specifically characteristic of osteomalacia. The defects start in the

cortex. Looser feels that the bone changes are brought about by slowly progressing callus formation, building up as small local infractions. These infractions he considers due to a "mechanical irritation due to strain."

Biopsy from the fracture sites are frequently difficult of interpretation as between normal callus and that formed in osteomalacia. Imperfect calcified bone matrix may be seen in the peripheral portions of the bone trabeculae. In well established cases the serum calcium, phosphorus and phosphatase levels are characteristic of osteomalacia. There is strong evidence that Milkman's disease is a form of osteomalacia and that treatment must vary with the etiologic factors to obtain the best treatment (Albright). Treatment includes vitamin D, a diet rich in calcium and phosphorus and correction of factors suc has renal acidosis, streatorrhea, etc.

Osteomalacia Due to Vitamin D Lack

1. *Fetal Rickets:* This disease is considered non-existent by many authors. Maxwell and also Snapper, however, report cases from China in which the mother was known to have osteomalacia at the time the affected children were born. The blood serum studies show a low calcium and normal or low phosphorus with an elevated phosphatase. The roentgenograms revealed osteoporotic changes in the bones. There was cupping and flaring of the epiphyseal ends of the long bones. None of the carpal bones were visible. Vitamin D therapy was quite effective in correcting the disease process and clearing up the bone deformities.

2. *Infantile Rickets* is a nutritional disturbance affecting the calcium and phosphorus balance and leading to marked bony changes in patients usually between four months and three years. It is found in children suffering from a deficiency in fat soluble vitamin D. The blood serum calcium and the serum phosphorus are lowered. The withdrawal of calcium from ossified skeletal tissue during active ossification removes the normal end point of ossification and leads to the proliferation of osteoblasts. This causes a rise in the blood alkaline phosphatase.

In rickets, as the spicules of bone are resorbed they are replaced by osteoid tissue which fails to calcify. The zone of cartilaginous proliferation is widened two to five times normal size and the alignment of cartilage is disturbed. Vascular spaces invade osteoid tissue in a desultory, irregular fashion. The vascular areolar spaces are filled by proliferating fibrous tissue. The zone of ossification is replaced by osteoid tissue without calcification.

The skeletal changes lead to a variety of deformities and defects

by roentgenogram. The most important diagnostic feature is a "saucer shaped" epiphyseal line, much widened and extremely irregular. Periosteal changes may occur with widening of the shaft; bowing of the tibiae may be extreme. The pelvis may be flattened or funneled and the skull may have a square contour. The affected bones have a definite thinned-out and osteoporotic appearance. Vitamin D, as well as vitamin C and a dietary rich in calcium are specific in the therapy of rickets.

3. *Late Rickets:* The few cases of rickets seen in older children past the age of three years have been designated as late rickets (Snapper). The fundamental bone changes are similar to those seen in early rickets. The cartilaginous plates increase in size; there is less evidence of the "calcification zone" and decalcification of bone trabeculae are found. Decalcification is definitely increased over that seen in infantile rickets.

4. *Adult Rickets:* This form of osteomalacia is relatively uncommon in the United States, but is still endemic in China and India, associated with pregnancy and lactation. The onset, between

Fig. 10-A. Roentgenogram of pelvis in adult rickets. Decalcification is moderate. Note marked deformity of pelvic bones. (Case of Dr. I. Snapper, reprinted from "Medical Clinics on Bone Diseases" by Dr. I. Snapper, Fig. A, Plate XI, Interscience Publishers, New York, N. Y., 1949.

the ages of 20 and 40 years, is usually insidious with vague indefinite pains in the back and groins. Progression, with motor disturbances, loose teeth, a waddly gait and, ultimately, inability to walk, occurs without treatment.

Roentgenologically, the bones show extreme decrease in density. The trabeculae are fine and the cortices are greatly thinned out. The long bones and spine show bizarre deformities; multiple cysts are infrequent. The pelvis may be much deformed (fig. 10A, B); pathologic fractures frequently occur.

Fig. 10-B. Note early changes in the cortex of the ulna, with discrete areas of absorption in the cortex, and with a mild generalized bone absorption of the ulna and radius. (Case of Dr. I. Snapper, reprinted from "Medical Clinics on Bone Diseases" by Dr. I. Snapper; Fig. E, Plate XI, Intersciences Publishers, Inc., New York, N. Y., 1949.)

The blood serum studies reveal a low or normal calcium level, a low serum phosphorus level and a high serum phosphatase level.

The treatment is based on the etiologic factors involved and includes diet, vitamins, calcium and, in the case of renal acidoses, alkalies.

B. *Renal Acidosis:* The renal osteodystrophies, associated with renal acidosis, include the following types and are associated with osteomalacic changes:

1. *"Tubular Insufficiency without Glomerular Insufficiency"* (Albright). In this condition there is inability of the tubules to secrete

an acid urine or form ammonia adequately, with consequent loss of calcium in the urine due to its substitution as a base. Lowering of serum calcium leads to parathyroid hyperplasia and, as the glomeruli are not affected, increased excretion of phosphates in the urine. Because of consequent lowering of serum phosphorus, calcium is not deposited in newly formed osteoid tissue.

2. *Fanconi Syndrome:* This condition seems to result from an abnormal excess of acid metabolites presented to the kidney for excretion. The normal compensatory mechanisms are overwhelmed, calcium is drawn from the serum to aid in the excretion of such metabolites and further changes occur as noted under Albright's syndrome above. The defect may be in the tubules and of such a nature as to inhibit normal reabsorption of amino acids although it is possible that there may be abnormal metabolism of amino acids elsewhere in the body. Disorders of cystine metabolism associated with bony lesions may fit into this syndrome.

The x-ray findings in both renal osteodystrophies are related to the lack of calcification of newly-formed osteoid tissue as opposed to the increased bone destruction seen in renal rickets, a form of osteitis fibrosa generalisata. The x-rays usually reveal some decalcification of all bones; there may be healed fractures of the long bones or phalanges, as well as the flat bones and, in many cases, there are changes consistent with long-standing rickets. Because of the fact that juvenile rickets has been largely controlled in this country these renal osteodystrophies and others are gaining in clinical significance.

The treatment consists in the administration of base, high calcium diet and vitamin D therapy. Once the bone lesions are controlled, alkali therapy alone will suffice.

II. Osteoporosity Related to Marked Bone Resorption

Primary Hyperparathyroidism with associated osteitis fibrosa generalisata may occur in juveniles or adults. This disease was first adequately described by von Recklinghausen in 1891. It is characterized by: hypercalcemia, hypercalcuria, hypophosphatemia, increased phosphatase content of the serum and increase in the phosphorus content of the urine.

There may be no skeletal changes visible by x-ray. In the majority of cases, however, varying amounts of rarefaction, deformities, cyst formation and fractures may be present (fig. 11A). The generalized demineralization of bone is a fundamental factor, because of the metabolic disturbance of calcium in the disease. The cysts may be extensive. The changes in the vertebra may be much

Fig. 11-A. Note the radiographic changes in the tibias and fibulas of a patient suffering from hyperparathyroidism. There are varying amounts of rarefaction and cyst formation. (Case of Dr. I. Snapper, reprinted from "Medical Clinics on Bone Diseases" by Dr. I. Snopper; Fig. B, Plate VII, Intersciences Publishers, Inc., New York, N. Y., 1949.)

like those seen in osteomalacia, multiple myeloma or metastatic carcinoma. Cystic tumors may occur in the jaws, the shafts of the long bones and in the ribs. Urinary calculi may be present. If the disease is arrested by the removal of the parathyroid tumor, the result symptomatically is dramatic. The osseous pain disappears, the cystic tumors in the bones, however, are healed in the space of from months to years but the abnormal blood serum findings are corrected quickly. There is frequently little left in the way of sequelae or findings.

When primary hyperparathyroidism from a parathyroid adenoma causes marked increase in calcium excretion, kidney damage may ultimately supervene. Because of the renal disease, acid metabolites are retained, creating the necessity for the use of calcium as a base with increased excretion of the calcium in the urine. This marked lowering of serum calcium results in parathyroid hyper-

plasia or secondary hyperparathyroidism, superimposed on the existing adenoma and the skeletal lesions become much more marked in severity.

Secondary Hyperparathyroidism with Renal Osteitis Fibrosa Cystica Generalisata

"Renal Rickets" is a disease of children and adolescents, a form of osteitis fibrosa generalisata. There is increased bone destruction; the kidneys show both glomerular and tubular insufficiency which may extend over a period of years. Retention of acid metabolites and inability of the kidneys to conserve base leads to increased excretion of calcium in the urine with lowering of serum calcium resulting in parathyroid hyperplasia and consequent fibrotic and cystic bone lesions. Because of phosphate retention, calcium complexes are normally deposited in newly-formed osteoid tissue, though at a slower rate than normal.

The radiologic findings may show the typical picture of infantile rickets with prominent changes at the diaphysical ends of the bones and a generalized osteoporosis. On the other hand, a second group may show lesions found in multiple osteitis fibrosa cystica. Deeply cupped metaphyses and subperiosteal erosion of the bone of the metaphyses may be present. The skull, roentgenographically, usually shows a moth-eaten, wooly and coarsely trabeculated appearance at the time other bones are showing decalcification. Occasionally, a picture resembling Paget's disease is found.

Treatment, as in the other renal dystrophies, consists of alkali, high calcium diet and vitamin D therapy.

Osteitis Deformans (*Paget's Disease*) is a disorder of the skeleton affecting primarily adults past the age of 45 years. The malacic changes associated with Paget's disease are usually found in the early stages of the disease, and it is at this stage that deformities usually result. As the bones harden, the deformities become fixed. Kyphosis is frequently found. Progressive enlargement of the head occurs and occasionally there is a change in its shape, the frontal area developing a square appearance. Pain in the lower extremities and in the vertebral column is frequent, when those regions are affected by the disease. The bones are characterized by marked resorption and by porous bone of a disordered architecture. The cortex may be thickened and the trabeculae may stand out prominently (fig. 12A). In the skull there is a marked thickening, at first of the outer table, with later involvement of the inner table. A coarse, mottled appearance is produced by dense, cotton-wool-like

Fig. 12-A. Typical radiographic changes in a case of Paget's disease, showing thickened cortex and prominent trabeculae in the tibia.

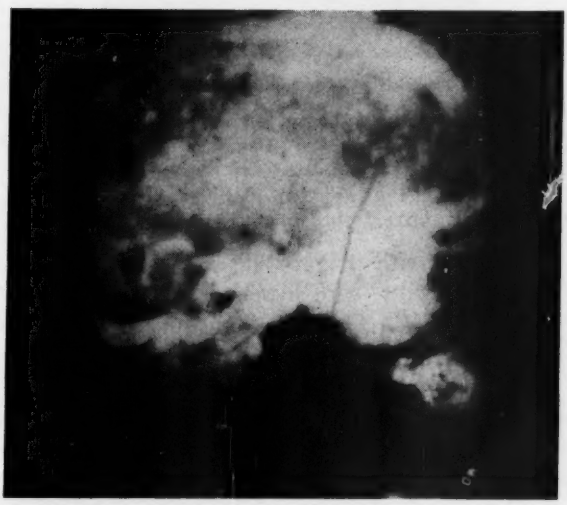

Fig. 12-B. Paget's disease of the skull with cotton-wool-like appearance. Note the areas of lessened density. There is thickening of the outer table of the skull.

areas, interspersed with areas of lessened density (fig. 12B). The sequence of events in Paget's disease seems to be a destruction of bone early in the disease, brought about by some unknown cause, followed by osteoblastic stimulation, with overgrowth of bone and an increase of the serum phosphatase level as a result of this osteoblastic stimulation. When one sees osteoporosis late in Paget's disease, there is reason to believe that it has been complicated by factors such as senile or menopausal osteoporosis (fig. 13). *The*

Fig. 13. A case of Paget's disease complicated by osteogenic sarcoma in the upper end of the femur, with an associated osteoporosis of the bones of the right extremity. (Case of Dr. Lawis G. Howard, Boston.)

background changes found in this disease are more like osteitis fibrosa cystica than those found in osteoporosis.

When a patient with Paget's disease is immobilized, acute atrophy of disuse results, with hypercalcinuria and an increase in geleralized osteoporosis. This reaction is much greater than in the normal individual. Indeed the possibility of a lethal outcome is possible. Treatment includes a diet high in calcium and phosphorus and vitamin D to increase calcium absorption. The patient is kept mobile if possible. On immobilization, as for a fracture or other reason, a low calcium and phosphorus diet with low vitamin D intake is essential. Large amounts of fluids should be given. Estrogenic therapy may be helpful in selected cases.

III. Osteoporotic or Malacic Changes Due to Other Diseases

Several of the numerous diseases, other than those already mentioned, which lead to malacic changes in bones will be mentioned briefly. They are: (a) Multiple myeloma; (b) Metastatic carcinoma; (c) Lymphosarcoma; (d) Hodgkin's disease; (e) the Leukemias; (f) Lipoid Granulomatosis of the bones; (g) Gaucher's disease; (h) Central defects, including (1) bone cyst; (2) Polyostotic fibrous dysplasia; (3) Monostatic fibrous dysplasia; (4) Neurofibromatosis; (i) Primary Tumors of bone or supporting framework, including: (1) Certain bone Sarcomas; (2) Fibrosarcoma; (3) Ewing's tumor; (4) Giant cell tumor; (5) Hemangioma.

With extensive osteoporosis, especially of the spinal column, the question of an osteoclastic type of metastatic lesion; a specific benign or malignant primary bone lesion; a primary lymphoma, etc., not infrequently must be considered.

IV. Comment

Diagnosis of the various entities described above is predicated upon a careful work-up of the case, including the history of trauma, pregnancy, lactation and the menopause. A dietary history considering the intake of calcium, vitamin D, and protein must be obtained. Adequate x-rays of the spine, pelvis, and long bones are frequently indicated. X-rays of the skull should be taken on all bone cases. Blood studies including calcium, phosphorus, alkaline phosphatase, non-protein nitrogen and carbon dioxide combining power, total proteins and formol-gel tests will frequently be helpful. Special tests, such as acid phosphatase, to rule out metastases from carcinoma of the prostate; urine for Bence-Jones protein studies; urine tests with Sulkowitch reagent; blood sedimentation tests and sternal puncture, all will yield significant findings under appropriate conditions. A routine urinalysis and blood count should never be omitted. Stool tests for fat and other abnormalities are to be done when indicated. In short, a careful survey of the patient with adequate laboratory studies and knowledge of the various causes of osteoporotic and malacic changes which occur should be of considerable benefit in colving many problems related to bone atrophy or destruction.

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LIGATION OF THE LARGER VENOUS CHANNELS*

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An increasing appreciation of the morbidity and mortality resulting from intravascular clot formation arouses considerable interest in any measure advanced for its management. This is certainly true in regard to the interruption of the larger venous channels.

Until relatively recent years vascular surgery was rather well limited to the treatment of aneurysms, pressure, cautery and ligation for trauma, and occasional daring attempts at embolectomy. Probably the first timid step toward present day advances was the treatment of varicose veins—by multiple ligations, stripping and then the deliberate production of intravascular clotting through the injection of sclerosing agents. An increased understanding of the physiology of the venous circulation was followed by the introduction of saphenous ligation. The advent of World War II with the large number of vascular injuries received in training as well as in combat presented many opportunities for appraisal of various procedures. Much was learned about the efficacy and importance of adequate collateral circulation. Blakemore tubes were introduced for the bridging and anastomosis of traumatized arteries. In common with other front-line installations we did not find them especially effective in combat wounds where the trauma to the collaterals was usually too extensive. They may, however, prove more satisfactory in civilian practice where only single tract bullet wounds and the ice-pick type of stab wound may present with good collaterals intact. What applies to the Blakemore tubes may also be said of vessel-grafts, shunts, etc.

Just before, during, and especially since the last war particular attention has been given to the study of phlebothrombosis, related or unrelated to surgery, thrombophlebitis, septic venous emboli and postphlebitic edema of the extremities. It is with these four types of cases that this presentation will concern itself in trying to evaluate the proper indications for ligation of the larger venous channels.

Phlebothrombosis

The popularizing of the term "phlebothrombosis" by Ochsner and his associates[1] has been of great value in enabling us to systematize our concept of the bland or silent type of thrombus whose

*Read by invitation before the first annual meeting of The Southwestern Surgical Assembly at Houston, Texas, Sept. 26 to 29, 1949.

floating end propagates in the blood stream and may easily become detached. In our consideration of this condition it is well to remember, however, that every case of intravascular clotting cannot be definitely catalogued as either phlebothrombosis or the more familiar thrombophlebitis. Both conditions may be present at one time, one may precede the other, or it may be that each represents a different phase of the same pathologic change. With an appreciation of those facts one cannot be too dogmatic in his discussion.

Generally speaking, the symptoms of phlebothrombosis, while they may be entirely absent, will usually manifest themselves with a cramp in the muscles of the calf of the leg or, less often, in the arch of the foot. This pain will likely be accompanied by tenderness in the corresponding area. If this tenderness is not palpable it may sometimes be elicited by Honan's sign—pain when the foot is dorsiflexed while the knee is slightly bent. Fever, increased pulse rate, and swelling will usually be minimal and not appear until after the pain or tenderness is observed. Contrary to the formerly held opinion that clot formation usually occurs in the pelvic veins it is most likely that the great majority begin in the deep veins of the calf which accounts for the symptoms just mentioned. Unfortunately in many instances pulmonary infarction may be the first indication of the presence of the lesion.

Early diagnosis is of extreme importance since the effectiveness of treatment may depend upon the time when it is instituted.

The former treatment of rest, ice applications, elevation and waiting for the expected embolus to be swept through the circulation has been rather generally abandoned. Instead of that the present day measures include even prophylactic procedures. Attempts are made to determine which cases are most likely to develop intravascular clotting, and early postoperative treatment is begun even before symptoms appear. Such treatment may include the use of pressure bandages, preoperative ligation of the superficial femoral veins, early postoperative activity, and the administration of anticoagulants.

Pressure bandages preoperatively and postoperatively, while rather favorably considered in European clinics, have not met with popular acceptance here. It must be admitted that they can relieve venous congestion and increase the force of venous flow. Possibly there is more value to this treatment than is generally recognized.

Unilateral or bilateral ligation of the superficial femoral veins is regularly employed prophylactively in selected cases at certain clinics. The usual indications are patients beyond the age of sixty,

and those requiring surgery for malignancy or for extensive procedures about the hip.

While there have been some conflicting reports regarding the value of early ambulation it seems rather certain that such activity will definitely not increase the incidence of clot formation but most likely will reduce it. It is my own conviction that Allen and his co-workers are correct in their belief that stasis is the outstanding etiologic factor.[2] One should, however, be explicit in what he means by early ambulation. It should not mean getting out of bed just to sit in a chair with flexion pressure on the femoral vessels at the groin and popliteal vessels at the knee. Rather it should mean what it says—the patient must walk. Otherwise he is much better off lying in bed with pillows under his knees but with encouragement to move his legs frequently—even to the extent of employing the bicycle pedaling type of exercise. Accept the fact that inactivity encourages thrombosis—then combat it as you will.

Unless contraindicated the prophylactic use of anticoagulants can be begun just a few hours postoperatively. If proper precautions are taken dicumarol or some form of heparin, alone or together, or some of the newer preparations can be rather safely used in an attempt to prevent thrombosis.

Once the presence of the lesion has been diagnosed active rather than expectant, treatment should be begun immediately. At present it will most likely consist of anticoagulant therapy or ligation of the larger veins.

Thrombophlebitis

In thrombophlebitis the danger of detachment of the clot is considerably less than in true phlebothrombosis but is still a factor, and a subsequent edema often presents a major problem for management.

Inflammation is the primary change in this disease and may be accompanied by various degrees of occlusion. The symptoms, as would be expected, are those of an inflammatory nature—redness, heat, swelling, tenderness, fever, chills, tachycardia, and malaise. In some cases the inflammation may be a peri-venous one and even resemble a lymphangitis.

Spasm of the arterial system undoubtedly causes much of the pain and other inflammatory symptoms. It is for this reason that sympathetic block is particularly applicable to this type of lesion. While it is frequently necessary to repeat the procedure a number of times, sometimes only one or two blocks will be necessary to break the chain of abnormal impulses carried through the reflex arch.

Anticoagulants are indicated to aid in limiting occlusion of the involved veins. Ligation of the larger veins is not generally practiced in the acute stage since massive killing emboli are rare. However, the smaller types of infarction are frequent and if they are often repeated or become more extensive may indicate the necessity for ligation.

Both phlebothrombosis and thrombophlebitis are so common that it does not seem necessary to present illustrative cases as will be given for septic venous emboli and chronic postphlebitic edema.

Septic Venous Emboli

Blood transfusion, special care, penicillin, sulfa compounds, streptomycin, aureomycin, etc., have been of such aid in the treatment of septicemia that we may fail to realize that at times this septic material may be incorporated in a thrombus and that septic venous emboli may result.

Not only may may the veins of the extremities be involved but the pelvic plexus, as a result of postabortal, puerperal or other pelvic infection, and also the prostatic plexus may be the origin of such emboli. If ligation becomes necessary it may be essential to ligate the inferior vena cava when either of the latter two are concerned. Another outstanding indication for such ligations is of course nonfatal pulmonary embolism.

Illustrative of the value of inferior vena caval ligation is the case of a patient who developed infection in the prostatic plexus following resection of the prostate gland. Septic emboli were released and on two occasions massive pulmonary infarcts occurred. The first one was severe enough—but in about five days another more extensive one developed. After competent consultation it was believed that the patient could not survive a third one and that such would probably occur. For that reason a ligation of the inferior vena cava was done under cyclopropane anesthesia using a right lateral extraperitoneal approach. Lumbar sympathectomy was not employed which may have accounted for the primary edema of the scrotum and extremities. This gradually subsided and while it later recurred it was believed to be due now to a hepatic cirrhosis unrelated to the operation itself. Certainly no more infarcts occurred and an apparently moribund patient survived eighteen months even with an advanced hepatic cirrhosis and ascites which eventually proved to be fatal.

Postphlebitic Edema

Once the storm of phlebothrombosis or especially thrombophlebitis has been weathered there may remain a more or less permanent

thickening or edema of the involved extremity. At best it is only unsightly, at worst it is heavy and uncomfortable or even disabling and the stretched skin may become the site of indolent ulcers resistant to all types of treatment previously used.

This edema has been treated by extremely prolonged bed rest followed by limited activity and various forms of supportive and often uncomfortable dressings.

In the past few years newer concepts of the pathological changes operating in such limbs have been offered.[3] It has been suggested that first the mechanical blocking of the vein and the destruction of its valves result in an increased venous pressure which in turn causes an increased filtration pressure and extravasation of fluid into the perivascular tissues. Accompanying this mechanical process detrimental impulses are sent from the diseased segment of vein through the sympathetic nerve fibers to the lumbar ganglia from which vasoconstrictor impulses are sent to both arteries and veins. The diminution in blood supply resulting from arteriospasm causes a tissue anoxia which increases the permeability of the capillary bed with additional loss of fluid into the tissue spaces.

The high venous pressure interferes with the passage of this fluid back into vascular channels and also lymph flow is retarded by inadequate pulsation of the arterioles.

Anticoagulants are of no value at this stage of the disease. A lumbar sympathectic block can, however, be expected to give some relief, but incomplete and temporary. Ligation and resection of the involved vein will accomplish the same thing and permanently by interrupting the afferent fibers carrying pain stimuli from the diseased vein. In addition it will shunt the venous blood through competent venous channels and further eliminate the rare possibility of emboli from below the point of ligation.

The extent of ligation which is permissible can be illustrated by one case where I did a bilateral ligation of the superficial femoral veins following a bilateral saphenous ligation which had been done elsewhere several years previously. This patient was frantic because of discomfort from the heaviness of the extremities and pain from repeated ulcers. It was felt that since the edema persisted after saphenous ligation that the femorals must be occluded and collateral circulation adequate. Such proved to be the case. While there is still considerable edema it is definitely less, both extremities are warmer, the old ulcers have healed and never ones are increasingly less frequent, smaller and more superficial.

Attention has been called also to the fact that unilateral edema

of the extremity in cases of pregnancy or pelvic tumors may be significant in that it may be the result of phlebothrombosis or thrombophlebitis, and not necessarily pressure alone.[4]

Anticoagulant Therapy

In phlebothrombosis, acute thrombophlebitis and septic emboli anticoagulant therapy has vigorous proponents and must be weighed against ligation. The use of anticoagulants is simpler, and seems to many to be less mutilating than the permanent ligation of large venous channels. If the prothrombin and coagulation time be controlled such treatment is not likely to do any additional damage to the body economy even if it does fail to prevent dissemination of emboli.

In every instance before anticoagulant therapy is used it must be remembered that there are certain definite contraindications. These include purpura and other blood dyscrasias with a tendency to bleeding, hepatogenous jaundice, renal insufficiency, ulcerative lesions, and recent operations upon the brain and cord.

The usual anticoagulants employed are dicumarol and heparin—alone or combined. A newer form of heparin in a gelatin and glucose base promises great possibilities and could establish itself as the ideal anticoagulant without the use of other drugs. Other preparations, still largely experimental, may prove to be even more effective. The actual technic of anticoagulant therapy does not properly fall within the scope of the presentation. If the results obtained are too drastic the drug should be stopped, large doses of vitamin K should be given and fresh whole blood transfusions administered. Should emboli occur while the patient is receiving anticoagulants ligation must be considered as an additional or supplementary form of treatment.

Ligation Therapy

While I am not ready now to follow the enthusiasts in their ever-widening indications for ligation, this treatment seems to be based upon sound reasoning and will probably be more and more generally adopted.

Ligation certainly does something that no other treatment will in that emboli cannot pass a resected ligated area of vein. Further, such ligation and resection will permanently prevent the carrying of abnormal impulses which often result in arteriospasm and its sequellae. Then, too, it will avoid any of the dangers of a decreased prothrombin time or prolonged coagulation time and eliminate the necessity for their frequent laboratory determination.

While distal clots are blocked by ligation it must in all fairness be emphasized that ligation has certain inadequacies. Proximal clots may require removal at the time of ligation, subsequently emboli may arise proximally but generally are small and not fatal,[5] and veins in the opposite extremity frequently develop the same process.

Ligation of the larger venous channels should carry a negligible mortality per se, and apparently such ligation does no harm to the venous circulation. This would indicate an overabundance of collateral venous channels and substantiate the view that the larger venous channels are principally reservoirs and not necessarily conduits. Even after ligation of the inferior vena cava there is no evidence of decrease of functional capacity, no claudication and no permanent edema. In this latter instance the collaterals used are probably the veins of the abdominal wall, the pelvis, retroperitoneal space and hemorrhoidal plexus.[6]

The level of ligation will depend upon the level of involvement of the diseased vein. If only the veins of the calf of the leg are involved ligation of the superficial femoral should be done. If cyanosis and edema indicate that clots extend into the common femoral it should be ligated. If the process extends into the iliac the common iliac should be ligated. However, because of the difficult approach, especially of the left side, some practice opening the common femoral and aspirating the clot in the iliac before ligating the common femoral. Others prefer ligation of the inferior vena cava in such instances and also when there is bilateral involvement of the common iliacs or femorals. As a general rule ligation proximal to the thrombus is cleaner and more desirable than lower ligation with attempted aspiration of the clot.

There are two points especially applicable to ligation of the inferior vena cava. One is that many believe a sympathectomy should be included and the other is that in postabortal or postpartum thrombosis with septic emboli the ovarian veins should be ligated also.[4]

The actual technic of ligation is not especially difficult. The most commonly ligated vein is the superficial femoral. While its designation as superficial is somewhat of a misnomer it can be readily exposed under local anesthesia through a longitudinal incision along the front of the thigh. Gentleness should be practiced in dissecting it from the artery in order to avoid setting up spasm. The procedure is of course incomplete without resection between the double ligature.

The common femoral is more difficult to expose. Ligation should

always be done close to the profunda branch. It should be remembered too that fewer collaterals are available for this level.

Because of the overlying sigmoid the left common iliac is so difficult to expose that many prefer ligature of the inferior vena cava especially since a bilateral common iliac ligation will often be necessary.

The inferior vena cava may be approached retroperitoneally or if a sympathectomy is to be done the transperitoneal route may be preferred. In either instance the lumbar veins must be carefully avoided to eliminate troublesome bleeding.

SUMMARY

In the foregoing discussion various explanations and types of treatment have been presented. The final answer to many of the questions is not yet settled.

It is suggested that among the topics requiring further study are the proper sphere for sympathetic block, the contrasting benefits of ligation versus anticoagulants in suspected phlebothrombosis, the advisability of preoperative femoral ligation in the aged, the desirability of bilateral femoral ligation for unilateral thrombosis, the prophylactic value of early ambulation, the determination of the proper level for ligation, the effectiveness of venous ligation in postphlebitic edema, the necessity for sympathectomy with inferior vena caval ligation and, not to be forgotten, the possible ill effects of any or all of the procedures considered.

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The Southern Surgeon

Published Monthly by

The SOUTHERN SURGEON PUBLISHING COMPANY

701 Hurt Building

ATLANTA 3

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Subscription in the United States, $8.00

Volume XVI | July, 1950 | Number 7

THE SURGICAL TREATMENT OF PORTAL HYPERTENSION

Recently there has been renewed interest in the surgical treatment of portal hypertension. Several of the procedures, such as the Talma-Morrison operation, operations designed to drain the ascitic fluid into the urinary, subcutaneous or venous systems, and the sclerosis of esophageal varices have not withstood the test of time. However, splenectomy, portocaval or splenorenal shunts and resection of the esophagus and stomach from reports with short-term follow-up seem encouraging.

We have recently reviewed the mortality in cirrhosis associated with medical treatment in the Hermann and Jefferson Davis Hospitals in Houston, Texas, during the period between Jan. 1, 1944, to Jan. 1, 1949. Of 158 patients with cirrhosis, 82 or 51 per cent expired within the first year after the diagnosis was made, 32 patients were known to live more than one year, and 44 patients could not be contacted. These figures agree closely with those of Ratnoff and Patek, who found that 60 per cent of patients with cirrhosis expired within the first year. It is because of this high

mortality associated with medical therapy that one is stimulated to seek other means of treatment. The early good results and low mortalities (10 to 15 per cent) with venous shunts reported by Linton, Blakemore, Whipple, Blalock, and our own experiences with these procedures, seem encouraging, in view of the poor results with present medical therapy.

ELLIOTT B. HAY, M.D.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

THE ABNORMAL PNEUMOENCEPHALOGRAM. By LEO M. DAVIDOFF, M.D., and BERNARD S. EPSTEIN, M.D. 506 pages, with 659 illustrations. Price, $15. Philadelphia: Lea & Febiger, 1950.

This book is a companion volume to "The Normal Encephalogram" which appeared in 1946. It was delayed by the death of Dr. Dyke, the co-author of that volume, and by the war.

The first section concerns neoplasms, the second non-neoplastic tumors and the final section covers non-tumorous lesions of the brain.

The arrangement of material under each subject includes a general discussion, review of the literature, a resume of the authors' own material, including review of plain roentgenograms and pneumoencephalograms, and a summary. Illustrations are well reproduced, those of the gross and microscopic specimens being especially excellent.

The book is easy to read and the correlation of the clinical, pathologic and roentgenologic aspects of diseases of the brain make it one many general practitioners, as well as radiologists and specialists in neurological fields, will find a valuable reference work.

LEONARD LONG, M.D.

VARICOSE VEINS. R. ROWDEN FOOTE. The C. V. Mosdy Company, St. Louis, Missouri. United States, 1949, 226 pages. $8.00.

This is the first volume in British Medical literature written solely on the subject of varicose veins. The volume begins with some historical remarks followed by the anatomy, physiology and pathology of veins. This volume covers not only the treatment of varicosities, but also thrombophlebitis, pulmonary embolism and includes a chapter on anti-coagulant therapy. The work covered is quite thorough, the writing is typically British, it is clear and concise.

The paper used is too thin, the printing of one side being impressed upon the other, there are many blemishes in the volume due to smudges from printers' ink, thus making the book rather difficult to read. The illustrations are fairly good.

If the printing and paper of this volume were of better quality, one would advise its place in the library of the general surgeon. Otherwise, it is quite worthwhile.

A. H. LETTON, M.D.

SURGERY FOR NURSES. JAMES MORONEY, M.D. The Williams and Wilkins Company. Baltimore: 1950. 644 pages. $6.00.

This volume, which was written by the examiner in surgery to the General Nursing Council for England and Wales, is rather inclusive, touching minorly on the many different aspects of surgery. The writing is typically English, as

are the illustrations. The illustrations are fairly good; many of them, however, are rather ancient.

The book is written primarily for students who are reviewing for the state examination, and as such the author spends lots of time on the unimportant things and leaves little explanation of the common, everyday disease and the problems met in the nursing profession. The instruments pictured here are those used in England and vary considerably from those popular in America at the present time.

This volume is probably of value for nurses studying for the state board examinations, but is not thorough enough nor is it applicable to our hospitals to be of use in the nurses' training school.

A. H. LETTON, M.D.

ACUTE INJURIES OF THE HEAD: *Their diagnosis, treatment, complications, and sequels.* By G. F. ROWBOTHAM, F.R.C.S. (Eng.). Third edition, 480 pp., with 259 illustrations. Baltimore: The Williams and Wilkins Company, 1949. $7.00.

This competent exposition by one skilled, by virtue of wide experience, in the management of head injuries is a welcome addition to the library of the general practitioner as well as to that of the specialist. It provides a readable medium for the critical analysis and adequate treatment of our daily increasing flood of head trauma resulting from traffic accidents. The author scientifically approaches the subject with his discussion of the mechanism, pathologic-physiology, as well as the practical application of diagnosis, treatment, and the often neglected discussion of rehabilitation and end results of this form of trauma. The bibliography is conveniently placed on each page which eliminates the usual search for references in books of this type. The illustrations are excellent, with the exception of minor criticism of the x-rays being positive reproductions rather than the regularly developed films which are employed for interpretation in everyday use. The use of immediate x-ray examination as an adjunct in the critical evaluation of the patient, and particularly the use of portable films, is probably not a suggestion that would meet with widespread approval. Other than these minor criticisms, the book is excellent and probably one of the best present-day treatises on this subject.

ROBERT F. MABON, M.D.

THE MERCK MANUAL OF DIAGNOSIS AND THERAPY, Eighth Edition, Published by MERCK AND COMPANY, INC., Rahway, N. J.

This manual contains 1592 pages and is thumb indexed. The thumb index contains the letters which refer to the code letter into which the contents have been divided in Part I. Part II and Part III are also indicated in the thumb index.

Part I is devoted to diseases and major symptoms, and many new or revised chapters on nutritional deficiencies, drug addiction, prenatal and postnatal care and the care of premature infants. Tropical and other exotic diseases of increasing significance, and summaries of anticoagulants, sulfonamides, antibiotic therapy and of treatment with adrenocortical steroids, etc., are discussed in Part I.

Part II devotes new chapters on routine immunization measures, clinical

and bedside procedures, laboratory tests, an outline on preoperative and postoperative care, a section on diets, ready reference data and conversion tables.

Part III is a complete index indicated by "Ind" on the thumb index. This manual contains a storehouse of valuable and almost indispensable information, *easy to find.*

BRANCHIOGENIC ANOMALIES. Results of Seventy Cases Observed at the Johns Hopkins Hospital between 1926 and 1946. GRANT E. BARD, JAMES W. HENDRICK and ROBERT G. CHAMBERS. *Western Journal of Surgery, Obstetrics and Gynecology 57*:536-549 (Nov.) 1949.

A variety of anomalies of the neck attributable to faulty development of the cephalic portion of the embryo may appear as fistulae, cysts, sinuses, ducts or solid cell rests. The most common are the branchial and thyroglossal abnormalities. This paper is concerned with those which the authors group loosely under the head of branchiogenic anomalies. These lesions are classified under three heads: (1) Fistulae, (2) Cysts and Sinuses, and (3) Carcinoma.

A brief presentation of the embryological anatomy concerned is followed by a discussion of the various theories as to the origin of these lesions. The authors feel that all that can be said with certainty is that these anomalies have some relation to the branchial apparatus.

Of the 70 cases in this series, 48 were cysts and 16 were fistulae. Seven (10 per cent) were branchiogenic carcinomas. One was a cylindroma. In one case both a cyst and a fistula were present. The diagnosis and accepted treatment of these lesions is presented. The differential diagnosis is discussed.

The authors feel that all branchiogenic anomalies should be excised because the ever-present possibility of carcinogenic change in embryologic rests.

ABSTRACTS FROM CURRENT LITERATURE

INCISION FOR EXPOSURE OF THE GALLBLADDER. John Spencer Carman, *J. Internat. Coll. Surgeons 13*:300-305 (March) 1950.

The author makes no claims for originality of this "physiologic" incision but states that it is an adaption at a particular site of recognized principles used elsewhere. The technic described for making this incision is quoted:

"Cutaneous Incision. A curvilinear incision starts from just above the right costal margin at a point immediately over the palpable gallbladder, or, if the organ cannot be felt, at a point where the fundus of the gallbladder is expected to lie. (This is usually between 1 and 2 inches (2.5 and 5 cm.) to the right of the junction of the lateral border of the right rectus muscle with the costal margin.) The incision then curves downward and across the abdomen, passing about an inch (2.5 cm.) above the umbilicus and ending about the same distance to the left of the midline, the latter part of the incision being horizontally transverse.

"External Oblique Aponeorosis. This layer is split in the direction of its fibers from the costal margin downward and medially, and this splitting is continued across the anterior rectus sheath to the midline, still following the direction of the external oblique fibers in that sheath.

"Internal Oblique Muscle and Sheath. These are cut across in the same direction as the splitting of the external oblique fibers.

"Rectus Abdominis Muscle. Whether the anterior sheath has been cut across obliquely or transversely will depend on the width of the upper part of the abdomen and, therefore, on the direction of the external oblique fibers. In any case, however, the right rectus abdominis muscle is cut transversely. This is best done with a scalpel, cutting across with a series of shallow cuts while the left index finger elevates the muscle away from the posterior sheath. This allows pressure to be exerted on the inferior epigastric artery (which may be multiple rather than single) and involves very slight loss of blood while the artery is being doubly clamped cut and ligated. (Note: There is frequently also a small artery at the outer border of the rectus sheath which needs to be clamped.)

"Posterior Rectus Sheath, Transversalis Muscle and Fascia and Peritoneam. It is most convenient to incise these structures together, in the same line as the rest of the incision."

A detailed description of the method of retraction and exposure employed as well as the author's suggestions for closure of the wound. He recommends a trial of this incision and report of experiences with it. In his hands it has been most satisfactory.

R. H. S.

TRANSGASTRIC PANCREATOCYSTOGASTROSTOMY. Ralph E. Scovel and Victor H. Holliger, *J. Internat. Coll. Surgeons 13*:278-284 (March) 1950.

Following a brief discussion of the pathogenesis, anatomic considerations, and diagnosis of pancreatic cysts, the authors suggest the following simple classification:

A. Benign cysts

1. Pseudocysts (most common)

2. Retention cysts (rare)
3. Congenital cysts (rare)
4. Parasitic cysts (very rare)

B. Neoplastic cysts (all very rare)
1. Cystadenoma
2. Cystadenocarcinoma
3. Teratoma

In consideration of the various methods of treatment, the authors are of the opinion that excision should be reserved for the neoplastic cysts and that marsupialization should be used only when circumstances offer no alternative. Internal drainage by cystovisceral anastomosis is considered to be the treatment of choice for the vast majority of pancreatic cysts. They consider transgastric pancreatocystogastrostomy to be preferable type of cystovisceral anastomosis. Their description of the technic of this procedure is quoted:

"Under suitable anesthesia, the abdomen is opened. The abdominal cavity is explored. If the transgastric approach is indicated as is usually the case, guy sutures are placed in the midportion of the anterior wall of the stomach. A transverse incision is made, and the guy sutures are used as retractors. Owing to the anterior displacement of the stomach by the cyst, the posterior wall of the stomach presents itself in the opening. The cyst is palpated through the gastrostomy wound and the site for the second opening is selected. Four guy sutures are placed through and through the posterior wall of the stomach and the adherent cyst. A large trocar is thrust through this area, and the cyst is aspirated. (The material may be saved for cell study.)

"The trocar is withdrawn and, with tension on the guy sutures, the opening is enlarged to about 4 cm. A palating index finger is now inserted and the wall of the cyst cavity carefully explored. A biopsy specimen, if advisable, may be obtained at this time. (If the rare malignant cyst is diagnosed, the feasibility of radical excision is considered. If the risk is deemed too great or the lesion considered inoperable, internal drainage, as stated, becomes the palliative procedure of choice.) If benignity is assured, the cystogastrostomy opening is ringed with interrupted gastrointestinal sutures to insure hemostasis. The possible purse-stringing effect of the continuous suture is avoided. Next the anterior gastrostomy wound is closed and the laparotomy is carried on to completion. No special postoperative care is needed other than Wangensteen suction for a day or so."

The report of the case of a 36 year old white man with a large pseudocyst successfully treated by this method is included.

R. H. S.

OSTEOID OSTEOMA: ETIOLOGY AND PATHOGENESIS. Report of Twelve New Cases. Bernard Pines, Leroy Lavine and David M. Grayzel, *J. Internat. Coll. Surgeons 13*:249-277 (March) 1950.

The purpose of this communication is to describe the clinical, roentgenologic and pathologic features of 12 new cases of osteoid osteoma. On the basis of this study and a review of the literature, a discussion of the controversial issues of etiology and pathogenesis is presented.

Clinically, osteoid osteoma is characterized by localized pain. It is variously described as sharp, dull, aching, boring, lancinating, burning, twitching, throb-

bing, drawing and digging in character. It is intermittent at the outset and later becomes more constant. It is, on occasion, worse at night. The pain is relieved by aspirin in practically all instances. Later, local signs of swelling and tenderness may appear but local redness is not a feature of the disease. Stiffness, contracture and deformity may occur. The duration of the complaints prior to admission to the hospital varies from a few months to a few years. The disease is one of adolescence and early adult life. Most patients are between the ages of 10 and 25 years. It occurs predominantly in males. A history of trauma was elicited in 30 to 40 per cent of all cases reported. (In the past, this relationship has not been considered to be of significance but the authors feel that this high instance of association should be taken into consideration.)

The most valuable aid in diagnosis is the roentgenographic study of the affected bone. Characteristically, the nidus appears as a small oval or round, sharply circumscribed area, radiolucent or (less frequently) radiopaque. At times the nidus is enveloped by a thin radiolucent circle. This in turn is surrounded by a denser area representing sclerotic reaction of adjacent bone. The x-ray evidence is not always conclusive. In most instances, the first impression of the roentgenologist was that of chronic inflammation.

The disease is infrequent but not rare. Since 1935 when Jaffe accurately described the entity and reported his five cases, about 150 cases have been reported. The authors add 12 new cases to this number. Illustrative x-ray studies and photomicrographs accompany the case reports. The reproduction of the x-ray plates is, unfortunately, poor but the photomicrographs are very good and point up some interesting observations on the frequency with which elements of chronic inflammation are noted in the sections.

A detailed discussion of the question of etiology is presented. Prior to 1935, the disease was thought to be inflammatory but Jaffe concluded that it was a benign new growth and suggested the term osteoma. Since that time various opinions have been offered. The authors feel that though the evidence is inconclusive, the weight of evidence appears to be in favor of the lesion representing a phase of chronic inflammation. They propose that if this be conclusively proved by future study, the term "osteoid osteitis" be used to designate this condition.

R. H. S.

UNDESCENDED TESTES. W. H. Snyder, Jr., William Van Valin and Lawrence Chaffin, *California Med. 72*:239-242 (April) 1950.

Orchiopexy was performed upon 152 patients. Follow-up observation was not altogether adequate but the authors consider that poor results were obtained in 14 per cent of the cases which could be followed.

Since an increasing number of investigators have observed spontaneous descent of undescended testes, it is felt that this possibility should always be considered in weighing the indications for operation on prepubescent boys. Coexisting clinically demonstrable hernia (59 per cent in this series) is considered to be a definite indication for early operation. Otherwise operation may be delayed until about the twelfth year. In the authors' series, this was done in 25 per cent of the cases.

Of the three methods employed, the greatest number of poor results were obtained with the Bevan technic (simple placement of the testicle in the

scrotum). Next in line was the Cabot-Nesbit procedure (traction suture through the testicle attached to the thigh). The fewest poor results were obtained with the Torek technic (direct suture of the testicle to the deep fascia of the thigh).

R. H. S.

Rectosigmoid Anastomosis by Invagination in Chronic Inflammatory Conditions. William H. Prioleau, *J. Internat. Coll. Surgeons 13*:388-393 (April) 1950.

"In spite of its long history and its prominent advocates, anastomosis of the rectum to the sigmoid by invagination without accurate suturing has gained little popularity." When used as a primary form of anastomosis, it carries a high mortality rate. Anastomosis by invagination is particularly indicated when chronic inflammatory changes prevent satisfactory mobilization of the rectum and when inflammatory disease makes it impossible to secure sufficiently pliable bowel ends to permit accurate approximation. "When the procedure is used under these conditions, it is most important that the site of anastomosis be protected, preferably by a preliminary transverse colostomy or by an accompanying tube cecostomy and intestinal intubation.

The author presents three cases in which this procedure has been used successfully. The technic of the procedure as described here is quoted:

"At operation the colon is mobilized sufficiently to permit its end to reach the rectum. This may be difficult because of extensive adhesions and because it has been shortened by resection of its diseased portion. It may be necessary to free the splenic flexure. The rectal stump is located by palpating the end of a tube that has been passed through the anal orifice. The end of the tube is drawn into the abdominal cavity through an incised opening made into the rectum, just large enough to permit a snug fit of the end of the sigmoid. No rectal mucosa is excised. The rectal tube is passed about 8 cm. into the sigmoid and secured against slipping and leakage by a heavy encircling transfixation suture. By traction upon the rectal tube, the sigmoid, with mesentery attached, is drawn into the rectum and a section of reasonably intact bowel is engaged for a length of several centimeters. The proximal end of the rectal tube should be above the rectal tube, which is prevented from slipping by securing a gauze sponge around it just outside of the anal orifice. The tube is kept open by irrigation and permits the passage of gas and liquid feces. The sutures holding it to the sigmoid slough out, and it may be withdrawn or expelled about the seventh day. After its removal, there is commonly some constriction, probably due to edema of the invaginated portion of the sigmoid. This is overcome by dilatation. Once an adequate lumen has been established, there is no tendency to reformation of stricture."

R. H. S.

Complete Abdominal Hysterectomy. A Simplified Technique and End Results in 500 Cases. Albert H. Alredge and Richard S. Meredith, *Am. J. Obst. & Gynec. 59*:748-759 (April) 1950.

"The advantages of removing the cervix as well as the body of the uterus in women who have indications for hysterectomy are that:

"1. Troublesome postoperative pelvic symptoms caused by chronic cervicitis are eliminated.

"2. An occasional unsuspected early carcinoma of the cervix is removed.

"3. The risk of subsequent development of cervical malignancy is prevented.

"4. Postoperative menopause symptoms are relatively less frequent and less severe than after the subtotal operation and patients adjust more readily to such symptoms when they do occur."

Te Linde has reported the incidental removal of four unsuspected carcinomas in a series of 300 cases in which total hysterectomy was performed. The author states that at the Woman's Hospital, 6 to 7 per cent of all cervical cancers occur in cervical stumps following subtotal hysterectomy. In the author's series of 500 cases, three unsuspected carcinomas of the cervix and one of the corpus were detected on pathologic examination of the specimen.

Convinced of the advantages of subtotal hysterectomy, the authors have tried to arrive at the most logical technic for the operation. It seemed obvious that such a procedure must be one that:

"1. Was technically as simple as possible.

"2. Would afford maximum protection against injury to the bladder, ureters and rectum.

"3. Could be done with a minimum of trauma to structures about the vaginal vault and to their blood supply.

"4. Would leave the vagina normal as to depth and the vaginal vault well supported and elastic.

"5. Was simple enough to be taught to members of our resident staff."

The technic which is described in detail and which was used in the 500 cases is a combination of features previously described and the authors make no claim to originality. Steps in this technic which have contributed to the safety of the procedure (the mortality rate for the series was 0.4 per cent) may be summarized as follows:

"1. Release of the cervix from the vault of the vagina by dissection within the pericervical cuff, thereby avoiding any risk of injury to the ureters, bladder or bowel.

"2. Effective prevention of hemorrhage by ligation of blood vessels in easily accessible safe areas.

"3. Promotion of wound healing by a minimum of trauma to structures about the vaginal vault and to their blood supply.

"4. Fixation of the detached proximal ends of the transverse cervical ligaments to the vaginal vault in a way which leaves it well supported and elastic.

"5. Elimination of all clamps from the pelvis to facilitate closure of the vaginal vault and peritonization of raw surfaces.

"6. Support of retained ovaries near the lateral walls of the pelvis to protect their blood supply and to prevent pelvic pain and dyspareunia from prolapse of the ovaries into the cul-de-sac."

Following analysis of the results in these cases (495 cases were followed), Aldridge and Meredith conclude:

"1. We have now techniques for complete removal of the uterus by the abdominal route which in the hands of competent surgeons are as safe as those for the subtotal operation.

"2. At least four out of five cervices removed from women who have indications for hysterectomy will show birth injuries, chronic inflammation, or an occasional unsuspected malignant growth.

"3. Complete removal of the uterus as a nearly routine procedure may prevent later development of an occasional carcinoma of the cervix.

"4. The incidence of postoperative pelvic complaints and the frequency and the severity of menopause symptoms are less after complete removal of the uterus than after subtotal hysterectomy.

"5. It is logical to choose the subtotal operation for some women who are definitely poor surgical risks.

"6. Results from utilization of the technique just described by a group of gynecologists and hospital residents, in a series of 500 cases, have convinced us that it is a simple, safe method for complete hysterectomy. Our experience has demonstrated that it can be readily adapted to varying conditions encountered in patients who have indications for removal of part or all of the uterus."

R. H. S.

AN ATTEMPT TO EVALUATE THE RADICAL AND PALLIATIVE TREATMENT OF BREAST CARCINOMA. Thomas G. Orr, *Surg., Gynec. & Obst. 90*:413-422 (April) 1950.

Though the author feels that strict adherence to the rigid criteria of operability of breast carcinoma as set forth by Haagensen and Stout may deny a small percentage of patients the possibility of cure, this difference of opinion does not detract from the value of these rules when applied to the teaching of young surgeons. On the contrary, young surgeons would do well to apply these rules and follow them with a few exceptions until further advancement is made in the treatment of breast cancer.

Denying that there is a standard technic for radical mastectomy and pleading for flexible adaption to the individual case, the writer offers his opinion on certain moot points. He feels that the skin incision should be adapted to the individual case and that local recurrence is probably representative of a failure to remove the disease from the axilla rather than insufficient resection of the skin. Removal of the clavicular portion of the pectoralis major muscle is of little importance and could scarcely affect the success of the procedure. "Since excision of the rectus fascia requires but a few moments and does not increase the hazard or mutilation of the operation, this fascia may, with good judgment, be removed routinely." Though the thoracodorsal nerve may be sacrified if there is evidence of metastasis along its course, the long thoracic nerve may be preserved without fear of altering the result of the procedure. In the writer's opinion, the advice of Trimble in regard to resection of the axillary vein only when it exhibits evidence of involvement in the disease process is sound. He feels that painstaking dissection of the axilla is more important than all the foregoing points.

In an analysis of the results of treatment in 315 traced cases, superior results obtained by a subradical procedure is explained on the basis of selection of cases for the modified procedure. The overall results in the Group I and II cases in this series are outstandingly good.

The author's conclusions on the subject are quoted:

"All types of therapy for carcinoma of the breast, except surgical, must be considered palliative. Since no one knows the percentage of cures that may be expected from surgical therapy, the term 'survivals' is more in keeping with our present knowledge of the natural history of the disease.

"If we accept irradiation as valuable treatment for inoperable cancer of the

breast, it would be illogical to assume that such treatment would be without merit as an adjunct to surgical therapy.

"Palliative results of castration and hormone therapy have been of sufficient value in the management of advanced carcinoma of the breast to justify their use in selected cases.

"Possibly more radical surgery in the future may salvage a very small percentage of breast carcinomas now considered incurable.

"It is evident that there are many problems relating to the treatment of carcinoma of the breast which remain unsolved. To champion one method of treatment or technic of operation to the exclusion of all others may smack of dogmatism, and dogmatism never does much toward the solving of scientific problems. Opinions should be respected, but should not be mistaken for facts."

R. H. S.

TREATMENT OF TRAUMATIC HERNIA OF THE DIAPHRAGM. Conrad R. Lam, *Arch. Surg. 60:*421-430 (March) 1950.

In considering the fact that in a ten year period, acute traumatic hernia of the diaphragm was diagnosed only three times at the Henry Ford Hospital (in the same period, 206 fractures of the pelvis were indexed), the author concludes that the diaphragm is a hardy structure and is remarkably resistant to disruption. The three acute hernias were operated on immediately but, even so, one suffered the complication of perforation of the incarcerated stomach. Although it would appear that prompt surgical repair is the treatment of choice, two elderly patients reported on here had received other advice and had been obliged to tolerate the presence of lengths of gastrointestinal tract in their troracic cavities for periods of several years.

Following the presentation of the three acute cases and the two cases in which injury had occurred at a much earlier date, the author presents a detailed account of the operative technic which he employs. With a Levine tube in the stomach and with endotracheal anesthesia, an incision is made over the seventh interspace. The chest is opened and exposure is maintained with self-retaining retractors. If diaphragmatic motion interferes with the reduction, he does not hesitate to crush the phrenic nerve. In recent injuries, reduction is easy but in long-standing cases, sharp dissection may be needed to separate the herniated viscera from the edges of the hernial opening. The diaphragmatic defect is repaired with a one-layer closure of heavy silk interrupted sutures. The diaphragm is sutured in the direction which affords the least tension. Gastric suction is employed for the first few postoperative days. Prophylactic penicillin is given and early ambulation is encouraged.

R. H. S.

CHOLEDOCHOSTOMY—ADVANTAGES OF A MODIFIED T TUBE. R. Russell Best, *Surg., Gynec. & Obst. 90:*295-297 (March) 1950.

For routine postoperative use or delayed cholangiographic studies, the long-used T tube with the longitudinal and transverse tubes attached at a 90 degree angle is satisfactory. When common duct stones or debris remain and the surgeon desires to take advantage of the nonoperative management for their dissolution, this arrangement has its disadvantages. Therefore, consideration was given to designing a tube with a 135 degree angle which would direct the flow of the injected solution toward the distal end of the duct. The desirability

of a double lumen arrangement led to attempts to catheterize the distal segment of the tube but this proved to be difficult. A double lumen tube was the obvious answer.

A 135 degree angle tube with a smaller lumen tube in the wall of the external and the distal segments is described. During the past year the author has had considerable experience with this tube and recommends it enthusiastically. When a cholangiogram is made, the contrast medium is introduced through the larger lumen and spreads both upward and downward. The smaller tube must be clamped during this procedure. When an antispasmodic is instilled to any solution for fragmentation, dissolution or irrigation is used, it is injected through the smaller opening. Any gas developing from the injected solution escapes through the larger lumen without the development of undue pressure.

The author suggests the use of this tube in all cases in which it is necessary to intubate the common duct.

R. H. S.

ANEURYSM OF THE SPLENIC ARTERY. Benjamin Sherwin and Harry Gordimer, *Ann. Surg. 131*:599-603 (April) 1950.

Approximately 144 cases of this rare entity have been reported. The authors add two others. The first case is that of a 60 year old woman who complained of vague upper abdominal pain radiating to the back and on x-ray examination of the abdomen was found to have a calcified density in the region of the splenic artery. Surgical resection of the spleen together with the artery and its calcified aneurysm was followed by uneventful recovery. The second case was also a 60 year old woman who was admitted with abdominal pain which had begun in the left upper quadrant and which was accompanied by vomiting. She had had gallbladder colic in the past. X-ray examination revealed a gallstone in the region of the gallbladder. An indefinite mass was present in the left upper quadrant which seemed to vary in size. She ran a moderate fever. About two weeks after admission, she had a sudden gastrointestinal hemorrhage but rallied after transfusion. Again after about two weeks, she had another gastrointestinal hemorrhage which proved fatal. At autopsy, the splenic artery exhibited four separate aneurysms, the most distal of which was ruptured. The large retroperitoneal hemorrhage had perforated secondarily into the stomach and duodenum.

The etiology is arteriosclerosis in most cases. Syphilis is not a factor. Mycotic aneurysms of this vessel are sometimes associated with rheumatic heart disease. It is of interest that at least 15 cases have been reported in which rupture of such an aneurysm complicated pregnancy.

The diagnosis is usually made at autopsy or occasionally at operation. The importance of making the diagnosis of splenic artery aneurysm, before rupture, and of operation to prevent the tragedy of rupture is emphasized. Since a bruit may be present in certain cases, the value of auscultation of the abdomen is mentioned.

Treatment consists of removal of the spleen together with the aneurysm, or if that is not feasible, proximal ligation of the splenic artery.

R. H. S.

THE REPAIR OF SINGLE CLEFT LIP BY THE HAGEDORN-LE MESURIER

TECHNIQUE. Claire L. Straith, Matthew A. Pilling and John R. Lewis, *J. Internat. Coll. Surgeons 4*:394-401 (April) 1950.

The authors describe a new harelip technic which they credit Hagedorn as describing in 1892 and credit Le Mesurier for reviving the method recently and popularizing it. This technic has been used with equal success in complete and incomplete clefts and for secondary repairs. The advantages are as follows: 1) It avoids the necessity of heavy skin sutures, 2) produces a more normal philtrum border, 3) eliminates an off-center scar, 4) equalizes the thickness of the mucous membrane, and 5) results in a more normal cupid's bow.

The authors recommend that cleft lips be repaired at the earliest possible age, the baby's health permitting, because the babies tolerate the operation well, the parents are spared prolonged heartache and embarrassment, and the repaired lip helps in the molding and shaping of the upper jaw, particularly when clefts are present. Preoperative preparation by mild sedation is described and local anesthesia is advised in newborn infants.

The technic is described in detail and results in a flap of skin and mucous membrane from the lateral side of the cleft which is turned downward and pulled medially to fill the defect created on the medial side of the cleft. This results in an incision passing inferiorly from the base of the repaired nostril down to within 1/8 of an inch of the vermilion border then medially to the midline, then downward again through the mucous membrane in the midline. Figure-of-eight sutures of 0000 nylon are introduced from beneath the lip to relieve tension and are tied under the lip. The skin edges are closed by interrupted sutures of very fine silk or nylon. An elipse of mucous membrane is excised from the under surface of the shorter side of the lip to equalize the thickness of the two sides. A logan traction bow is applied to relieve further tension on the suture line. Early removal of skin sutures and proper cleansing of the suture line help to insure an insignificant scar.

The Hagedorn-Le Mesurier technic has been used exclusively by the authors for the past three years and has in their opinion given consistently better results than other methods. Before and after photographs of various types of unilateral cleft lips are shown in babies and adults and an example of a secondary repair is also shown to prove further the merits of this technic.

R. H. S.

PERIPHERAL ARTERIAL EMBOLISM. William DeWitt Andrus, *Arch. Surg. 60*:511-519 (March) 1950.

Embolism of the arteries of the extremities is a dramatic event which varies in gravity and consequence. Embolism of an artery such as the posterior tibial, radial or even the brachial or axillary may be attended by no symptoms other than transient pain and weakness. Sudden embolism of the larger vessels of the lower extremity is of graver significance but even here the prognosis depends upon many things other than the local vascular occlusion. For example, the prognosis is much better in a patient with rheumatic heart disease than in a patient whose cardiac disease is of arteriosclerotic origin. Not only is this true in conservatively managed cases but such factors profoundly affect the results following embolectomy.

Spasm of other vessels of the limb and propagation of the clot may seriously impair the collateral circulation. Thus in all cases of embolism involving the

extremities, the most effective methods of combating the associated spasm should be employed at once. These procedures include interruption of the sympathetic pathways (either by sympathetectomy or by paravertebral block) which should be done whether or not embolectomy has been performed, and the employment of such drugs as papaverine and more recently, tetraethylammonium. Mechanical measures such as alternate positive and negative pressure have been used in the past. Further propagation of the clot should be combated with heparin or dicumarol.

The cardiac status of the patient is highly important since in failure, the "head of pressure" in the arterial circulation may be lowered, and increased venous pressure, which may further impair circulation of the part, may be present.

The present study was undertaken with the idea of re-evaluating the relative values of conservative management and embolectomy. The case material includes 15 cases of saddle embolus, involving one or both iliac arteries and 57 other instances of embolus in the arteries of the extremities.

Of the ten cases of saddle embolus, six were seen so late in the course of serious heart failure as to make any treatment ineffective, and in another instance, the embolism occurred in the late months of pregnancy in a 38 year old woman with rheumatic heart disease. All seven died. The remaining three survived but developed gangrene in one leg and were treated with mid-thigh amputation. Embolectomy was performed in one who survived and in one who died.

The 57 instances of embolism of the arteries of the extremities occurred in 50 patients. All were treated conservatively and none had embolectomy. Sixteen of these patients died and of these five suffered their embolism so late in the course of cardiac disease that any form of treatment was useless. Two of this group of sixteen died of subacute bacterial endocarditis five to thirty days following the occlusion of the artery. The mortality rate of those with arteriosclerotic heart disease was 52.2 per cent as contrasted with 13 per cent in those with rheumatic heart disease.

After reviewing these statistics, the author concludes that saddle embolus, embolectomy should be performed if the condition of the patient will allow it. While the results have been good with conservative management of other types of peripheral embolism, he feels that they could be improved and that embolectomy should be used in some instances, particularly in the younger age group.

Treatment of the syndrome should fall into three general categories: a) Measures aimed at relief of the spasm; b) anticoagulant therapy; and c) Embolectomy in those patients who have saddle embolus and will tolerate the procedure, in those of the younger age group, and in those with femoral or politeal embolism seen within twelve hours of onset when the process is due to an old rheumatic heart disease which is compensated.

R. H. S.